This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

Harvard University

Library of

The Medical School

and

The School of Public Health

The Gift of

J.G. Greenfield, M.D.

8136
2-5D

MEDICO-CHIRURGICAL TRANSACTIONS.

PUBLISHED BY

THE ROYAL

MEDICAL AND CHIRURGICAL SOCIETY

OF

LONDON.

VOLUME THE SIXTY-SEVENTH.

LONDON:
LONGMANS, GREEN, AND CO.,
PATERNOSTER ROW.

1884.

HARVARD UNIVERSITY
SCHOOL OF MEDICINE AND PUBLIC HEALTH
LIBRARY

29 NOV 1935
Gift: Dr J.G. Greenfield
G.G.143(1)

MEDICO-CHIRURGICAL TRANSACTIONS.

PUBLISHED BY

THE ROYAL

MEDICAL AND CHIRURGICAL SOCIETY

OF

LONDON.

SECOND SERIES.

VOLUME THE FORTY-NINTH.

LONDON:

LONGMANS, GREEN, AND CO.,

PATERNOSTER ROW.

1884.

PRINTED BY J. E. ADLARD, BARTHOLOMEW CLOSE.

ROYAL
MEDICAL AND CHIRURGICAL SOCIETY
OF LONDON.

PATRON.

THE QUEEN.

OFFICERS AND COUNCIL,
ELECTED MARCH 1, 1884.

President.

GEORGE JOHNSON, M.D., F.R.S.

VICE-PRESIDENTS.	WILLIAM OVEREND PRIESTLEY, M.D. REGINALD EDWARD THOMPSON, M.D. WILLIAM SCOVELL SAVORY, F.R.S. RICHARD BARWELL.
TREASURERS.	CHARLES BLAND RADCLIFFE, M.D. JOHN COOPER FORSTER.
SECRETARIES.	RICHARD DOUGLAS POWELL, M.D. MATTHEW BERKELEY HILL.
LIBRARIANS.	WILSON FOX, M.D., F.R.S. JOHN WHITAKER HULKE, F.R.S.
OTHER MEMBERS OF COUNCIL.	GEORGE FIELDING BLANDFORD, M.D. DYCE DUCKWORTH, M.D. SAMUEL JONES GEE, M.D. FREDERICK WILLIAM PAVY, M.D. CHARLES THEODORE WILLIAMS, M.D. JOHN CROFT. SIR WILLIAM MAC CORMAC. THOMAS PICKERING PICK. WILLIAM SEDGWICK. WILLIAM SPENCER WATSON.

THE ABOVE FORM THE COUNCIL.

RESIDENT LIBRARIAN.

JAMES BLAKE BAILEY.

A LIST OF THE PRESIDENTS OF THE SOCIETY FROM ITS FORMATION.

ELECTED

1805. WILLIAM SAUNDERS, M.D.
1808. MATTHEW BAILLIE, M.D.
1810. SIR HENRY HALFORD, Bart., M.D., G.C.H.
1813. SIR GILBERT BLANE, Bart., M.D.
1815. HENRY CLINE.
1817. WILLIAM BABINGTON, M.D.
1819. SIR ASTLEY PASTON COOPER, Bart., K.C.H., D.C.L.
1821. JOHN COOKE, M.D.
1823. JOHN ABERNETHY.
1825. GEORGE BIRKBECK, M.D.
1827. BENJAMIN TRAVERS.
1829. PETER MARK ROGET, M.D.
1831. SIR WILLIAM LAWRENCE, Bart.
1833. JOHN ELLIOTSON, M.D.
1835. HENRY EARLE.
1837. RICHARD BRIGHT, M.D., D.C.L.
1839. SIR BENJAMIN COLLINS BRODIE, Bart., D.C L.
1841. ROBERT WILLIAMS, M.D.
1843. EDWARD STANLEY.
1845. WILLIAM FREDERICK CHAMBERS, M.D., K.C H.
1847. JAMES MONCRIEFF ARNOTT.
1849. THOMAS ADDISON, M.D.
1851. JOSEPH HODGSON.
1853. JAMES COPLAND, M.D.
1855. CÆSAR HENRY HAWKINS.
1857. SIR CHARLES LOCOCK, Bart., M.D.
1859. FREDERIC CARPENTER SKEY.
1861. BENJAMIN GUY BABINGTON, M.D.
1863. RICHARD PARTRIDGE.
1865. SIR JAMES ALDERSON, M.D., D.C.L.
1867. SAMUEL SOLLY.
1869. SIR GEORGE BURROWS, Bart., M.D., D.C.L.
1871. THOMAS BLIZARD CURLING.
1873. CHARLES JAMES BLASIUS WILLIAMS, M.D.
1875. SIR JAMES PAGET, Bart., D.C.L., LL.D.
1877. CHARLES WEST, M.D.
1879. JOHN ERIC ERICHSEN.
1881. ANDREW WHYTE BARCLAY, M.D.
1882. JOHN MARSHALL.
1884. GEORGE JOHNSON, M.D.

FELLOWS OF THE SOCIETY APPOINTED BY THE COUNCIL AS REFEREES OF PAPERS.

FOR THE SESSION OF 1884-85.

BAXTER, EVAN BUCHANAN, M.D.
BECK, MARCUS.
BELLAMY, EDWARD.
BROADBENT, WILLIAM HENRY, M.D.
BRUNTON, THOMAS LAUDER, M.D., F.R.S.
BRYANT, THOMAS.
CREIGHTON, CHARLES, M.D.
CURNOW, JOHN, M.D.
DICKINSON, WILLIAM HOWSHIP, M.D.
DUNCAN, JAS. MATTHEWS, M.D., LL.D., F.R.S.
ERICHSEN, JOHN E., LL.D., F.R.S.
FAYRER, SIR JOSEPH, K.C.S.I., M.D., F.R.S.
FENWICK, SAMUEL, M.D.
GALABIN, ALFRED LEWIS, M.D.
GERVIS, HENRY, M.D.
GREEN, T. HENRY, M.D.
HARLEY, JOHN, M.D.
HEWITT, GRAILY, M.D.
HOLMES, TIMOTHY, M.A.
HUTCHINSON, JONATHAN, F.R.S.
LEGG, JOHN WICKHAM, M.D.
LOWNE, BENJAMIN THOMPSON.
MACNAMARA, CHARLES.
MARCET, WILLIAM, M.D., F.R.S.
MORRIS, HENRY.
MOXON, WALTER, M.D.
ORD, WILLIAM MILLER, M.D.
PAGE, HERBERT WILLIAM, M.A., M.C.
POLLOCK, GEORGE DAVID.
SMITH, THOMAS.
STURGES, OCTAVIUS, M.D.
WARING, EDWARD JOHN, M.D.
WEBER, HERMANN, M.D.
WILLETT, ALFRED.
WILLIAMS, JOHN, M.D.
WOOD, JOHN, F.R.S.

TRUSTEES OF THE SOCIETY.

SIR GEORGE BURROWS, Bart., M.D., D.C.L., F.R.S.
THOMAS BLIZARD CURLING, F.R.S.

TRUSTEES OF THE MARSHALL HALL MEMORIAL FUND.

WALTER BUTLER CHEADLE, M.D.
WILLIAM OGLE, M.D.
THOMAS SMITH.

LIBRARY COMMITTEE FOR THE SESSION OF 1884-85.

THOMAS LAUDER BRUNTON, M.D., F.R.S.
ALFRED LEWIS GALABIN, M.A., M.D.
WILLIAM R. GOWERS, M.D.
J. WICKHAM LEGG, M.D.
JOSEPH FRANK PAYNE, M.D.
MARCUS BECK, C.M.
FREDERICK JAMES GANT.
J. WARRINGTON HAWARD.
JEREMIAH MacCARTHY, M.A.
WILLIAM JOHNSON WALSHAM, C.M.

Hon. Secs. { R. DOUGLAS POWELL, M.D.
M. BERKELEY HILL.

Hon. Libs. { WILSON FOX, M.D., F.R.S.
JOHN WHITAKER HULKE, F.R.S.

FELLOWS

OF THE

ROYAL MEDICAL AND CHIRURGICAL SOCIETY

OF LONDON.

EXPLANATION OF THE ABBREVIATIONS.

P.—President.	V.P.—Vice-President.
T.—Treasurer.	S.—Secretary.
L.—Librarian.	C.—Member of Council.

The figures succeeding the words *Trans.* and *Pro.* show the number of Papers which have been contributed to the Transactions or Proceedings by the Fellow to whose name they are annexed. *Referee, Sci. Com.*, and *Lib. Com.*, with the dates of office, are attached to the names of those who have served on the Committees of the Society.

OCTOBER, 1884.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents those marked thus (*) are entitled by composition to receive the Transactions.

Elected

1846 *ABERCROMBIE, JOHN, M.D.

1877 ABERCROMBIE, JOHN, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; 39, Welbeck street, Cavendish square.

1851 *ACLAND, SIR HENRY WENTWORTH, K.C.B., M.D., LL.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Regius Professor of Medicine in the University of Oxford; Broad Street, Oxford.

1847 ACOSTA, ELISHA, M.D., 24, Rue de Luxembourg, St. Honoré, Paris.

Elected

1852 †ADAMS, WILLIAM, Surgeon to the Great Northern Hospital and to the National Hospital for the Paralysed and Epileptic; Consulting Surgeon to the National Orthopædic Hospital, Great Portland street; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 3.

1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde park.

1837 *AINSWORTH, RALPH FAWSETT, M.D., Consulting Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.

1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.

1866 ALLBUTT, THOMAS CLIFFORD, A.M., M.D., F.R.S., Physician to the Leeds General Infirmary; 35, Park square, Leeds. *Trans.* 3.

1879 ALLCHIN, WILLIAM HENRY, M.B., Physician to, and Lecturer on Medicine at, the Westminster Hospital; 5, Chandos street, Cavendish square, W.

1863 ALTHAUS, JULIUS, M.D., Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's park; 48, Harley street, Cavendish square. *Trans.* 2.

1884 ANDERSON, ALEXANDER RICHARD, Resident Surgeon, General Hospital, Nottingham.

1881 ANDERSON, JAMES, A.M., M.D., 84, Wimpole street, Cavendish square.

1862 ANDREW, EDWYN, M.D., 12, St. John's Hill, Shrewsbury.

1862 ANDREW, JAMES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 22, Harley street, Cavendish square. S. 1878-9. C. 1881-2. *Trans.* 1.

1820 ANDREWS, THOMAS, M.D., Norfolk, Virginia.

1880 *APPLETON, HENRY, M.D., Staines.

1819 †ARNOTT, JAMES MONCRIEFF, F.R.S., Chapel House, Lady Bank, Fifeshire, and 36, Sussex gardens, Hyde park. L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6. P. 1847-8. *Referee*, 1842-45, 1849-54, 1857-64. *Lib. Com.* 1836. *Trans.* 8.

Elected

1874 AVELING, JAMES H., M.D., Physician to the Chelsea Hospital for Women; 1, Upper Wimpole street, Cavendish square.

1851 *BAKER, ALFRED, Consulting Surgeon to the Birmingham General Hospital; 3, Waterloo street, Birmingham.

1873 *BAKER, J. WRIGHT, Senior Surgeon to the Derbyshire General Infirmary; 102, Friargate, Derby.

1865 BAKER, WILLIAM MORRANT, Surgeon to, and Lecturer on Physiology at, St. Bartholomew's Hospital; Consulting Surgeon to the Evelina Hospital for Sick Children; 26, Wimpole street, Cavendish square. C. 1878-9. *Lib. Com.* 1876-7. *Trans.* 6.

1869 BAKEWELL, ROBERT HALL, M.D., Ross, Westland, New Zealand.

1839 †BALFOUR, THOMAS GRAHAM, M.D., F.R.S., Surgeon General; Coombe Lodge, Wimbledon Park. C. 1852-3. V.P. 1860-1. T. 1872. *Lib. Com.* 1849. *Trans.* 2.

1848 †BALLARD, EDWARD, M.D., Inspector, Medical Department, Local Government Board; 12, Highbury terrace, Islington. C. 1872. V.P. 1875-6. *Referee*, 1853-71. *Lib. Com.* 1855. *Trans.* 5.

1866 *BANKS, JOHN THOMAS, M.D., Physician in Ordinary to the Queen in Ireland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Regius Professor of Physic in the University of Dublin; Member of the Senate of the Queen's University in Ireland; 45, Merrion square, Dublin.

1879 BARKER, ARTHUR EDWARD JAMES, Assistant Surgeon to, and Assistant Professor of Clinical Surgery at, University College Hospital; 87, Harley street, Cavendish square. *Trans.* 3.

1882 BARKER, FREDERICK CHARLES, M.D., Surgeon-Major, Bombay Medical Service [care of ARTHUR E. J. BARKER, 87, Harley street].

Elected

1833 †BARKER, THOMAS ALFRED, M.D., Consulting Physician to St. Thomas's Hospital; 109, Gloucester place, Portman square. C. 1844-5. V.P. 1853-4. T. 1860-2. *Referee*, 1848-51. *Trans.* 6.

1876 BARLOW, THOMAS, M.D., B.S., Assistant Physician to University College Hospital, to the Hospital for Sick Children, Great Ormond street, and to the London Fever Hospital; 10, Montague street, Russell square. *Trans.* 1.

1881 *BARNES, HENRY, M.D., F.R.S. Ed., Physician to the Cumberland Infirmary; 6, Portland square, Carlisle.

1861 BARNES, ROBERT, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; 15, Harley street, Cavendish square. C. 1877-8. *Referee*, 1867-76. *Lib. Com.* 1869-73. *Trans.* 4.

1864 BARRATT, JOSEPH GILLMAN, M.D., 8, Cleveland gardens, Bayswater.

1880 BARROW, A. BOYCE, Assistant Surgeon to the Westminster Hospital and to the West London Hospital; 17, Welbeck street, Cavendish square.

1840 BARROW, BENJAMIN, Surgeon to the Royal Isle of Wight Infirmary; Southlands, Ryde, Isle of Wight.

1859 BARWELL, RICHARD, *Vice-President*, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital; 32, George street, Hanover square. C. 1876-77. V.P. 1883-4. *Referee*, 1868-75, 1879-82. *Trans.* 9.

1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Pathological Anatomy in University College, London, and Physician to University College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 20, Queen Anne street, Cavendish square. *Trans.* 1.

1874 BAXTER, EVAN BUCHANAN, M.D., Professor of Materia Medica at King's College, London; Physician to King's College Hospital; 28, Weymouth street, Portland place. *Referee*, 1881-4.

Elected

1875 BEACH, FLETCHER, M.B., Medical Superintendent, Metropolitan District Asylum, Darenth, near Dartford, Kent.

1883 BEALE, EDWIN CLIFFORD, M.A., M.B., Assistant Physician to the City of London Hospital for Diseases of the Chest; and Physician to the Great Northern Hospital; 23, Upper Berkeley street.

1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-77. *Referee*, 1873-5. *Trans.* 1.

1860 *BEALEY, ADAM, M.D., M.A., Oak Lea, Harrogate.

1856 BEARDSLEY, AMOS, F.L.S., Bay villa, Grange-over-Sands, Lancashire.

1871 BECK, MARCUS, M.S., Teacher of Operative Surgery, and Professor of Clinical Surgery in University College, London; and Assistant Surgeon to University College Hospital; 30, Wimpole street, Cavendish square. *Referee*, 1882-4. *Lib. Com.* 1881-4.

1880 BEEVOR, CHARLES EDWARD, M.D., Assistant Physician to the National Hospital for the Paralysed and Epileptic; 33, Harley street, Cavendish square.

1858 BEGLEY, WILLIAM CHAPMAN, A.M., M.D., late of the Middlesex County Lunatic Asylum, Hanwell; 26, Saint Peter's square, Hammersmith. C. 1877–8.

1883 BELL, HUTCHINSON ROYES, Surgeon to, and Demonstrator of Operative Surgery at, King's College Hospital; 12, Queen Anne street, Cavendish square.

1871 BELLAMY, EDWARD, Surgeon to, and Lecturer on Anatomy at, Charing Cross Hospital; Lecturer on Artistic Anatomy to the Science and Art Department, South Kensington; 17, Wimpole street, Cavendish square. *Referee*, 1882-4. *Lib. Com.* 1879-81.

1847 BENNET, JAMES HENRY, M.D., The Ferns, Weybridge.

Elected

1880 BENNETT, ALEX. HUGHES, M.D., Assistant Physician to the Westminster Hospital; 38, Queen Anne street, Cavendish square, and 1, Petersham terrace, Gloucester road, South Kensington.

1883 BENNETT, STORER, 17, George street, Hanover square.

1877 BENNETT, WILLIAM HENRY, Assistant Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; Surgeon to the Belgrave Hospital for Children; 1, Chesterfield street, Mayfair.

1845 †BERRY, EDWARD UNWIN, 17, Sherriff road, West Hampstead.

1820 BERTIN, STEPHEN, Paris.

1872 BEVERLEY, MICHAEL, M.D., Assistant Surgeon to the Norfolk and Norwich Hospital; 63, St. Giles's street, Norwich.

1865 *BICKERSTETH, EDWARD ROBERT, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Liverpool Royal Infirmary School of Medicine; 2, Rodney street, Liverpool. *Trans.* 1.

1878 BINDON, WILLIAM JOHN VEREKER, M.D.

1854 BIRD, PETER HINCKES, F.L.S., Post Office, Limassol, Cyprus.

1856 BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.

1849 †BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square. C. 1865-6. *Referee*, 1851-9.

1851 †BIRKETT, JOHN, F.L.S., Consulting Surgeon to Guy's Hospital; Corresponding Member of the "Société de Chirurgie" of Paris; Inspector of Anatomy for the Provinces in England and Wales; 62, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866, 1869, *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.

1866 BISHOP, EDWARD, M.D., Cintra park, Upper Norwood.

Elected

1881 Biss, Cecil Yates, M.D., Assistant Physician to the Hospital for Consumption and to the Middlesex Hospital; 65, Harley street, Cavendish square. *Trans.* 1.

1865 Blanchet, Hilarion, Examiner to the College of Physicians and Surgeons, Lower Canada; 6, Palace street, Quebec, Canada east.

1865 Blandford, George Fielding, M.D., Lecturer on Psychological Medicine at St. George's Hospital; 71, Grosvenor street. C. 1883-4.

1867 Bloxam, John Astley, Surgeon to, and Teacher of Operative Surgery in, Charing Cross Hospital; Surgeon for Out-Patients to the Lock Hospital; Junior Surgeon to the West London Hospital; 8, George street, Hanover square.

1823 Bojanus, Louis Henry, M.D., Wilna.

1846 †Bostock, John Ashton, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-Major, Scots Fusilier Guards; 73, Onslow gardens, Brompton. C.1861-2. V.P. 1870-71. *Sci. Com.* 1867.

1869 Bourne, Walter, M.D. [care of the National Bank of India, 80, King William street, City.]

1882 Bowlby, Anthony A., Curator of the Museum, St. Bartholomew's Hospital, 75, Warrington crescent, Maida hill.

1870 *Bowles, Robert Leamon, M.D., 8, West terrace, Folkestone.

1841 †Bowman, Sir William, Bart., LL.D., F.R.S., F.L.S., Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Referee*, 1845-50, 1854-6. *Lib. Com.* 1847. *Trans.* 3.

1884 Boyd, Stanley, M.B., Assistant Surgeon to the Charing Cross Hospital; 62, Guilford street, Russell square.

1862 Brace, William Henry, M.D., 7, Queen's Gate terrace, Kensington.

Elected

1874 BRADSHAW, A. F., Surgeon-Major; Surgeon to the Rt. Hon. the Commander in Chief in India; Army Head Quarters, Bengal Presidency. [Agent: Vesey W. Holt, 17, Whitehall place.]

1883 BRADSHAW, JAMES DIXON, M.B., 30, George Street, Hanover square.

1867 *BRETT, ALFRED T., M.D., Watford, Herts.

1876 BRIDGES, ROBERT, M.B., Manor House, Yattendon, Newbury, Berks.

1867 BRIDGEWATER, THOMAS, M.B., Harrow-on-the-Hill, Middlesex.

1868 BROADBENT, WILLIAM HENRY, M.D., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the London Fever Hospital; 34, Seymour street, Portman square. *Referee*, 1881-4. *Trans.* 5.

1851 †BRODHURST, BERNARD EDWARD, F.L.S., Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor street. C. 1868-9. *Lib. Com.* 1862-3. *Trans.* 2. *Pro.* 1.

1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 8, Chesterfield street, Mayfair. *Trans.* 1.

1860 BROWN-SÉQUARD, CHARLES EDOUARD, M.D., LL.D., F.R.S., Laureate of the Academy of Sciences of Paris; Professor of Medicine at the College of France; Professor of General Physiology at the Museum of Natural History; Paris. *Sci. Com.* 1862.

1878 BROWNE, JAMES CRICHTON, M.D., LL.D., F.R.S., Lord Chancellor's Visitor in Lunacy; Lecturer on Mental Diseases, St. Mary's Hospital; 7, Cumberland Terrace, Regent's Park.

1880 BROWNE, JAMES WILLIAM, M.B., 8, Norland place, Uxbridge road.

1881 BROWNE, JOHN WALTON, M.D., Surgeon to the Belfast Ophthalmological Hospital; 10, College square N., Belfast.

1881 BROWNE, OSWALD A., M.A., St. Bartholomew's Hospital; 25, Bernard street, Russell square.

Elected

1874 BRUCE, JOHN MITCHELL, M.D., Physician to, and Lecturer on Materia Medica at, the Charing Cross Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 70, Harley street. *Trans.* 1.

1871 BRUNTON, THOMAS LAUDER, M.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, St. Bartholomew's Hospital; Examiner in Materia Medica in the University of London; 50, Welbeck street, Cavendish square. *Referee*, 1880-84. *Lib. Com.* 1882-4.

1860 BRYANT, THOMAS, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 53, Upper Brook street, Grosvenor square. C. 1873-4. *Sci. Com.* 1863. *Referee*, 1882-4. *Lib. Com.* 1868-71. *Trans.* 9. *Pro.* 1.

1855 BRYANT, WALTER JOHN, Physician to the Home for Incurable Children, Maida vale; 23A, Sussex square, Hyde park gardens.

1823 BUCHANAN, B. BARTLET, M.D.

1864 BUCHANAN, GEORGE, M.D., F.R.S., Medical Officer of the Local Government Board; Member of the Senate of the University of London; 24, Nottingham place, Marylebone road.

1864 BUCKLE, FLEETWOOD, M.D.

1876 BUCKNILL, JOHN CHARLES, M.D., F.R.S.; E 2, The Albany, Piccadilly, and Hill Morton Hall, Rugby.

1881 BULLER, AUDLEY CECIL, late City of London Hospital for Diseases of the Chest, Victoria Park.

1833 †BURROWS, SIR GEORGE, Bart., M.D., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen; Consulting Physician to St. Bartholomew's Hospital; Member of the Senate of the University of London; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V. P. 1849-50. P. 1869-70. *Referee*, 1842-6, 1850-7, 1861-68, 1875-81. *Lib. Com.* 1836. *Trans.* 2.

Elected

1837 †BUSK, GEORGE, F.R.S., F.L.S., Consulting Surgeon to the Seamen's Hospital, Greenwich; Member of the Senate of the University of London; 32, Harley street, Cavendish square. C. 1847-8. V.P. 1855. T. 1866. *Referee*, 1846-54, 1857-65. *Lib. Com.* 1847. *Trans.* 4.

1873 BUTLIN, HENRY TRENTHAM, Assistant Surgeon to, and Demonstrator of Practical Surgery and of Diseases of the Larynx at, St. Bartholomew's Hospital; 47, Queen Anne street, Cavendish square. *Trans.* 3.

1871 BUTT, WILLIAM F., 25, Park street, Park lane.

1883 BUXTON, DUDLEY WILMOT, M.D., B.S., 99, Gower Street.

1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 56, Grosvenor street, Grosvenor square.

1851 *CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 24, St. Giles's street, Norwich. *Trans.* 1.

1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place, Hyde park.

1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 27, Queen Anne street, Cavendish square. *Trans.* 1.

1845 †CARTWRIGHT, SAMUEL, late Professor of Dental Surgery at King's College, London, and Surgeon-Dentist to King's College Hospital; Consulting Surgeon to the Dental Hospital; 32, Old Burlington street. C. 1860-1. *Sci. Com.* 1863.

1879 CARTWRIGHT, S. HAMILTON, Professor of Dental Surgery at King's College; 32, Old Burlington street.

1868 CAVAFY, JOHN, M.D., Physician to St. George's Hospital; 2, Upper Berkeley street, Portman square. *Trans.* 1.

1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital; Physician to the London Fever Hospital and to the North-Eastern Hospital for Children; 27, Wimpole street, Cavendish square. *Trans.* 1.

Elected

1884 CHAFFEY, WAYLAND CHARLES, M.B., Medical Registrar to the Hospital for Sick Children, Great Ormond street; 28, Cedars road.

1845 †CHALK, WILLIAM OLIVER, 3, Nottingham terrace, York gate, Regent's park. C. 1872-3.

1844 †CHAMBERS, THOMAS KING, M.D., Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's Hospital and to the Lock Hospital; Shrubs Hill House, Sunningdale, Staines. C. 1861. V.P. 1867. L. 1869-72. *Referee*, 1851-60, 1866. *Lib. Com.* 1852, 1868. *Trans.* 1.

1879 CHAMPNEYS, FRANCIS HENRY, M.A., M.B., Assistant Lecturer on Midwifery, and Assistant Obstetric Physician to St. George's Hospital; 60, Great Cumberland place. *Trans.* 5.

1859 CHANCE, FRANK, M.D., Burleigh House, Sydenham Hill.

1849 CHAPMAN, FREDERICK, Old Friars, Richmond Green, Surrey.

1877 CHARLES, T. CRANSTOUN, M.D., Lecturer on Practical Physiology at St. Thomas's Hospital; Crofton Lodge, Hopton road, Coventry park, Streatham.

1881 *CHAVASSE, THOMAS FREDERICK, M.D., C.M., Surgeon to the Birmingham General Hospital; 24, Temple Row, Birmingham. *Trans.* 1.

1868 CHEADLE, WALTER BUTLER, M.D., Physician (with charge of out-patients) to, and Lecturer on Medicine at, St. Mary's Hospital; Physician to the Hospital for Sick Children; 2, Hyde park place, Cumberland gate.

1879 CHEYNE, WILLIAM WATSON, M.B., Assistant Surgeon and Demonstrator of Surgery to King's College Hospital; 14, Mandeville place, Manchester square, W.

1873 *CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.

Elected

1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Hospital, and to the Margaret Street Infirmary for Consumption; 63, Grosvenor street, Grosvenor square. C. 1881-2. *Referee*, 1873-80.

1872 CHRISTIE, THOMAS BEITH, M.D., Medical Superintendent, Royal India Asylum, Ealing.

1866 CHURCH, WILLIAM SELBY, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. *Referee*, 1874-81.

1860 CLARK, SIR ANDREW, Bart., M.D., LL.D., Physician to, and Lecturer on Medicine at, the London Hospital; 16, Cavendish square. C. 1875.

1879 CLARK, ANDREW, Assistant Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 19, Cavendish place, Cavendish square, W.

1839 †CLARK, FREDERICK LE GROS, F.R.S., Consulting Surgeon to St. Thomas's Hospital; The Thorns, Sevenoaks. S. 1847-9. V.P. 1855-6. *Referee*, 1859-81. *Lib. Com.* 1847. *Trans.* 5.

1882 CLARKE, ERNEST, M.B., B.S., 21, Lee terrace, Blackheath.

1848 †CLARKE, JOHN, M.D., 42, Hertford street, May Fair. C. 1866.

1881 CLARKE, W. BRUCE, M.B., Assistant Surgeon to, and Demonstrator of Anatomy at, St. Bartholomew's Hospital; 46, Harley street, Cavendish square.

1842 †CLAYTON, SIR OSCAR MOORE PASSEY, Extra Surgeon-in-Ordinary to H.R.H. the Prince of Wales, and Surgeon-in-Ordinary to H.R.H. the Duke of Edinburgh; 5, Harley street, Cavendish square. C. 1865.

1879 CLUTTON, HENRY HUGH, M.A., M.B., Assistant Surgeon to, and Lecturer on Forensic Medicine at, St. Thomas's Hospital; 2, Portland place.

1857 COATES, CHARLES, M.D., Consulting Physician to the Bath General and Royal United Hospitals; 10, Circus, Bath.

Elected

1868 COCKLE, JOHN, M.D., F.L.S., Physician to the Royal Free Hospital; 13, Spring gardens, Charing cross. *Trans.* 2.

1865 COOPER, ALFRED, Surgeon for Out-patients to the Lock Hospital; Assistant Surgeon to St. Mark's Hospital; Surgeon to the West London Hospital; 9, Henrietta street, Cavendish square.

1843 †COOPER, WILLIAM WHITE, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Ophthalmic Surgeon to St. Mary's Hospital; 19, Berkeley square. C. 1858-9. V.P. 1873-4. *Lib. Com.* 1847, 1856-7.

1868 CORNISH, WILLIAM ROBERT, Surgeon-Major, Madras Army; Sanitary Commissioner for Madras; Secretary to the Inspector-General, Indian Medical Department.

1860 *CORRY, THOMAS CHARLES STEUART, M.D., Ormean Terrace, Belfast.

1864 COULSON, WALTER JOHN, Surgeon to the Lock Hospital, 17, Harley street, Cavendish square.

1860 †COUPER, JOHN, Surgeon to the London Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital; 80, Grosvenor street. C. 1876. *Referee* 1882-3.

1877 COUPLAND, SIDNEY, M.D., Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; 14, Weymouth street, Portland place.

1862 COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon to the Victoria Hospital for Children; 3, Cavendish place, Cavendish square. C. 1882-3.

1841 CRAWFORD, MERVYN ARCHDALL NOTT, M.D., Millwood, Wilbury road, Brighton. C. 1853-4.

1868 CRAWFORD, THOMAS, M.D., Director General, Army Medical Department; 6, Whitehall yard, and 5, St. John's park, Blackheath.

1873 CREIGHTON, CHARLES, M.D., 6, Queen Anne street, Cavendish square. *Referee*, 1882-4. *Trans.* 1.

Elected

1869 *Cresswell, Pearson R., Dowlais, Merthyr Tydvil.

1874 Cripps, William Harrison, Assistant Surgeon to St. Bartholomew's Hospital; 2, Stratford place, Oxford street. *Trans.* 1.

1882 Crocker, Henry Radcliffe, M.D., Physician to the Skin Department, University College Hospital; Assistant Physician to the East London Hospital for Children; 28, Welbeck street, Cavendish square. *Trans.* 1.

1868 Croft, John, Surgeon to, and Lecturer on Clinical Surgery at, St. Thomas's Hospital; 48, Brook street, Grosvenor square. C. 1884. *Lib. Com.* 1877-8. *Trans.* 1.

1862 Crompton, Samuel, M.D., Brookmead, Cranleigh, Surrey.

1837 Crookes, John Farrar, 45, Augusta gardens, Folkestone.

1872 Crosse, Thomas William, Surgeon to the Norfolk and Norwich Hospital; 22, St. Giles's street, Norwich.

1849 *Crowfoot, William Edward, Beccles, Suffolk.

1879 Cumberbatch, A. Elkin, Aural Surgeon to St. Bartholomew's Hospital; Aural Surgeon to the Great Northern Hospital; 17, Queen Anne street.

1846 Curling, Henry, Consulting Surgeon to the Margate Royal Sea-Bathing Infirmary; Augusta Lodge, Ramsgate, Kent.

1837 †Curling, Thomas Blizard, F.R.S., Consulting-Surgeon to the London Hospital; 27, Brunswick square, Brighton. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. P. 1871-2. *Referee*, 1844-6, 1851-3, 1858, 1865-70, 1875-9. *Sci. Com.* 1863. *Lib. Com.* 1839. *Trans.* 13. *Pro.* 1.

1873 Curnow, John, M.D., Professor of Anatomy at King's College, London, and Physician to King's College Hospital; 3, George street, Hanover square. *Referee*, 1884.

1847 Currey, John Edmund, M.D., Lismore, County Waterford.

1822 Cusack, Christopher John, Chateau d'Eu, France.

Elected

1872 DALBY, WILLIAM BARTLETT, M.B., Lecturer on Aural Surgery at St. George's Hospital; 18, Savile row. *Trans.* 2.

1884 DALLAWAY, DENNIS, Langham Hotel.

1877 DARBISHIRE, SAMUEL DUKINFIELD, M.D., Physician to the Radcliffe Infirmary, Oxford; 60, High street, Oxford.

1879 DARWIN, FRANCIS, M.B., F.R.S., The Grove, Huntingdon road, Cambridge.

1848 DAUBENY, HENRY, M.D., San Remo, Italy.

1874 DAVIDSON, ALEXANDER, M.D., Physician to the Liverpool Northern Hospital; 2, Gambier terrace, Liverpool.

1853 DAVIES, ROBERT COKER NASH, Rye, Sussex.

1852 DAVIES, WILLIAM, M.D., 2, Marlborough buildings, Bath.

1876 DAVIES-COLLEY, J. NEVILLE C., M.C., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 36, Harley street, Cavendish square. *Trans.* 2.

1878 DAVY, RICHARD, F.R.S. Ed., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 33, Welbeck street, Cavendish square.

1882 DAWSON, YELVERTON, M.D., 28, Hyde park street.

1867 DAY, WILLIAM HENRY, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.

1878 DENT, CLINTON THOMAS, Assistant Surgeon to St. George's Hospital; 19, Savile row, Burlington gardens. *Trans.* 2.

1859 †DICKINSON, WILLIAM HOWSHIP, M.D., Physician to, and Lecturer on Medicine at St. George's Hospital, and Senior Physician to the Hospital for Sick Children; 9, Chesterfield street, Mayfair. C. 1874-5. *Referee*, 1869-73, 1882-4. *Sci. Com.* 1867-79. *Trans.* 13.

1839 †DIXON, JAMES, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Harrow Lands, Dorking. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Referee*, 1865. *Lib. Com.* 1845-8. *Trans.* 4.

Elected

1862 DOBELL, HORACE B., M.D., Consulting Physician to the Royal Hospital for Diseases of the Chest, City road; Streate place, Bournemouth. *Trans.* 2.

1845 DODD, JOHN.

1879 DONKIN, HORATIO, M.B., Physician to the Westminster Hospital; Physician to the East London Hospital for Children; 60, Upper Berkeley street, Portman square.

1877 DORAN, ALBAN HENRY GRIFFITHS, Assistant Surgeon to the Samaritan Free Hospital; 51, Seymour street, Portman square.

1863 DOWN, JOHN LANGDON HAYDON, M.D., Physician to, and Lecturer on Clinical Medicine at, the London Hospital; 81, Harley street, Cavendish square. C. 1880. *Trans.* 2.

1867 DRAGE, CHARLES, M.D., Hatfield, Herts.

1879 DREWITT, F. G. DAWTREY, M.D., Assistant Physician to the West London Hospital and to the Victoria Hospital for Children; 52, Brook street, Grosvenor square.

1884 DRUITT, LIONEL, M.D., 122, Clapham road.

1880 DRURY, CHARLES DENNIS HILL, M.D., Bondgate, Darlington.

1865 DRYSDALE, CHARLES ROBERT, M.D., Physician to the Farringdon Dispensary; Assistant-Physician to the Metropolitan Free Hospital; 97, Regent street.

1865 DUCKWORTH, DYCE, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. C. 1883-4. *Trans.* 1.

1876 DUDLEY, WILLIAM LEWIS, M.D., Physician to the City Dispensary; 149, Cromwell road, South Kensington.

1845 DUFF, GEORGE, M.D., High street, Elgin.

1874 DUFFIN, ALFRED BAYNARD, M.D., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 18, Devonshire street, Portland place.

1874 DUKA, THEODORE, M.D., [Surgeon-Major, H.M.'s Bengal Army].

Elected

1871 DUKE, BENJAMIN, 2, Windmill road, Clapham common.

1871 *DUKES, CLEMENT, M.D., B.S., Sunnyside, Rugby, Warwickshire.

1867 DUKES, M. CHARLES, M.D., Wellesley Villa, Wellesley road, Croydon.

1880 DUNBAR, JAMES JOHN MACWHIRTER, M.D., Hedingham House, Clapham common.

1877 DUNCAN, JAMES MATTHEWS, M.D., LL.D., F.R.S., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Bartholomew's Hospital; Examiner in Obstetric Medicine, University of London; 71, Brook street, Grosvenor square. *Referee*, 1881-4. *Trans.* 1.

1863 DURHAM, ARTHUR EDWARD, F.L.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 82, Brook street, Grosvenor square. C. 1876-7. *Referee*, 1880-1. *Sci. Com.* 1867. *Lib. Com.* 1872-5. *Trans.* 5.

1874 DURHAM, FREDERIC, M.B., 38, Brook street, Grosvenor square.

1843 DURRANT, CHRISTOPHER MERCER, M.D., Consulting Physician to the East Suffolk and Ipswich Hospital; Northgate street, Ipswich, Suffolk.

1872 EAGER, REGINALD, M.D., Northwoods, near Bristol.

1868 EASTES, GEORGE, M.B. Lond., Surgeon-Accoucheur to the Western General Dispensary; 69, Connaught street, Hyde park square.

1883 EDMUNDS, WALTER, M.D., 79, Lambeth Palace road, Albert Embankment.

1883 EDWARDES, EDWARD JOSHUA, M.D., 17, Orchard street, Portman Square, W.

1824 EDWARDS, GEORGE.

1823 EGERTON, CHARLES CHANDLER, Kendall Lodge, Epping.

1869 ELAM, CHARLES, M.D., Assistant-Physician to the National Hospital for the Paralysed and Epileptic; 75, Harley street, Cavendish square.

Elected

1848 ELLIS, GEORGE VINER, late Professor of Anatomy in University College, London; Minsterworth, Gloucester. C. 1863-4. *Trans.* 2.

1868 ELLIS, JAMES, M.D., the Sanatorium, Anaheim, Los Angeles County, California.

1854 *ELLISON, JAMES, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1842 †ERICHSEN, JOHN ERIC, LL.D.,F.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Surgery in University College, London, and Consulting Surgeon to University College Hospital; 6, Cavendish place, Cavendish sq. C. 1855-6. V.P. 1868. P. 1879-80. *Referee*, 1866-7, 1884. *Lib. Com.* 1844-7, 1854. *Trans.* 2.

1879 EVE, FREDERIC S., Pathological Curator of the Museum, Royal College of Surgeons; Assistant Surgeon, Royal Free Hospital; 7, Welbeck street, Cavendish square. *Trans.* 2.

1877 EWART, WILLIAM, M.D., Assistant Physician to, and Lecturer on Physiology at, St. George's Hospital; 33, Curzon street, Mayfair.

1875 *FAGAN, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, the Belfast Royal Hospital; 1, Glengall place, Belfast.

1869 FAIRBANK, FREDERICK ROYSTON, M.D., 46, Hallgate, Doncaster.

1862 FARQUHARSON, ROBERT, M.D., M.P., Finzean, Aboyne, Aberdeenshire, and the Reform Club, Pall Mall. *Lib. Com.* 1876-80.

1844 †FARRE, ARTHUR, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Physician-Accoucheur to H.R.H. the Princess of Wales; 18, Albert Mansions, Victoria street, Westminster. C. 1857. V.P. 1864. *Referee*, 1848-54, 1861-3, 1865-6. *Sci. Com.* 1863. *Lib. Com.* 1847.

Elected

1872 FAYRER, SIR JOSEPH, K.C.S.I., M.D., F.R.S., Honorary Physician to H.M. the Queen, and to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; Surgeon-General, late Bengal Medical Service; Examining Medical Officer to the Secretary of State for India in Council; President of the Indian Medical Board; 53, Wimpole street, Cavendish square. *Referee*, 1881-4.

1872 *FENWICK, JOHN C. J., M.D., Physician to the Durham County Hospital; 25, North road, Durham.

1863 FENWICK, SAMUEL, M.D., Physician to the London Hospital; 29, Harley street, Cavendish square. C. 1880. *Referee*, 1882-4. *Trans.* 4.

1880 FERRIER, DAVID, M.D., LL.D., F.R.S., Professor of Forensic Medicine at King's College, London, and Physician to King's College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 16, Upper Berkeley street, Portman square. *Trans.* 2.

1852 *FIELD, ALFRED GEORGE.

1849 †FINCHAM, GEORGE TUPMAN, M.D., Consulting Physician to the Westminster Hospital; 13, Belgrave road, Pimlico. C. 1871.

1879 FINLAY, DAVID WHITE, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, the Middlesex Hospital; Physician to the Royal Hospital for Diseases of the Chest; 21, Montagu street, Portman square.

1866 FISH, JOHN CROCKETT, B.A., M.D., Assistant Physician to the West London Hospital; 92, Wimpole street, Cavendish square.

1866 FITZPATRICK, THOMAS, M.D., M.A., Physician to the Western General Dispensary; 30, Sussex gardens, Hyde park.

1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Consulting Physician to the Birmingham General Hospital; 8, Clarendon crescent, Leamington. *Trans.* 1.

Elected

864 *FOLKER, WILLIAM HENRY, Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.

1877 FONMARTIN, HENRY DE, M.D., Parkhurst, Isle of Wight.

1848 †FORBES, JOHN GREGORY, Egerton House, Egerton, Ashford, Kent. C. 1868-9. *Lib. Com.* 1855. *Trans.* 2.

1852 †FORSTER, JOHN COOPER, *Treasurer*, Consulting Surgeon to the Royal Hospital for Children and Women; President of the Royal College of Surgeons; 29, Upper Grosvenor street. C. 1868-9. S. 1873-5. V.P. 1877-8. T. 1879-84. *Referee*, 1870-2, 1876. *Pro.* 1.

1877 *FORTESCUE, GEORGE, M.B., late Surgeon to the Sydney Infirmary; 6, Lyons terrace, Sydney, New South Wales.

1865 FOSTER, BALTHAZAR WALTER, M.D., Professor of Medicine at the Queen's College, Birmingham, and Physician to the Birmingham General Hospital; 14, Temple row, Birmingham.

1883 FOWLER, JAMES KINGSTON, M.A., M.D., Assistant Physician to the Middlesex Hospital, and to the Hospital for Consumption, Brompton; 35, Clarges street, Piccadilly.

1859 FOX, EDWARD LONG, M.D., Consulting Physician to the Bristol Royal Infirmary; Church House, Clifton, Gloucestershire.

1880 FOX, THOMAS COLCOTT, B.A., M.B., Physician to the Skin Department of the Paddington Green Hospital for Children, and Assistant Physician to the Victoria Hospital for Children; 14, Harley street, Cavendish square. *Trans.* 1.

1858 FOX, WILSON, M.D., F.R.S., *Librarian*, Physician-Extraordinary to H.M. the Queen; Physician in Ordinary to H.R.H. the Duke of Edinburgh; Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 67, Grosvenor street. C. 1875-6. L. 1883-4. *Referee*, 1869-74. *Lib. Com.* 1866-70, 1874. *Trans.* 3.

Elected

1871 FRANK, PHILIP, M.D., Cannes, France.

1843 FRASER, PATRICK, M.D. C. 1866.

1868 FREEMAN, WILLIAM HENRY, 21, St. George's square, South Belgravia.

1836 †FRENCH, JOHN GEORGE, 10, Cunningham place, St. John's Wood road. C. 1852-3.

1883 FULLER, HENRY ROXBURGH, M.B., 45, Curzon street, May Fair.

1876 FURNER, WILLOUGHBY, Assistant Surgeon to the Sussex County Hospital; 2, Brunswick place, Brighton.

1864 *GAIRDNER, WILLIAM TENNANT, M.D., LL.D., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Western Infirmary, Glasgow; 225, St. Vincent street, Glasgow.

1874 GALABIN, ALFRED LEWIS, M.A., M.D., Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; Assistant Physician to the Hospital for Sick Children; 49, Wimpole street, Cavendish square. *Referee*, 1882-4. *Lib. Com.* 1883-4. *Trans.* 2.

1883 GALTON, JOHN CHARLES, M.A., F.L.S., 45, Great Marlborough street.

1865 GANT, FREDERICK JAMES, Senior Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde park. C. 1880-81. *Lib. Com.* 1882-4. *Trans.* 3.

1867 GARLAND, EDWARD CHARLES, Yeovil, Somerset.

1867 GARLIKE, THOMAS W., Malvern Cottage, Churchfield road, Ealing.

1854 GARROD, ALFRED BARING, M.D., F.R.S., Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 8.

Elected

1879 GARSTANG, THOMAS WALTER HARROPP, Dobcross, near Oldham.

1851 †GASKOIN, GEORGE, Surgeon to the British Hospital for Diseases of the Skin; 7, Westbourne park. C. 1875-6. *Trans.* 2.

1819 GAULTER, HENRY.

1848 †GAY, JOHN, Senior Surgeon to the Great Northern Hospital, and Consulting Surgeon to the Asylum for Idiots; 51, Belsize Park, Hampstead.. C. 1874-5.

1866 GEE, SAMUEL JONES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; Physician to the Hospital for Sick Children; 54, Wimpole street, Cavendish square. C. 1883-4. *Sci. Com.* 1879. *Lib. Com.* 1871-6. *Trans.* 1.

1878 GERVIS, HENRY, M.D., Obstetric Physician to, and Lecturer on Obstetric Medicine at, St. Thomas's Hospital; Examiner in Obstetric Medicine at the University of London; 40, Harley street, Cavendish square. *Referee*, 1884.

1884 GIBBES, HENEAGE, M.D., Physician to the Metropolitan Dispensary; Lecturer on Morbid Histology, Westminster Hospital; 94, Gower street.

1880 GIBBONS, ROBERT ALEXANDER, M.D., Physician to the Grosvenor Hospital for Women and Children; 32, Cadogan place.

1877 GODLEE, RICKMAN JOHN, Assistant-Surgeon to University College Hospital; Surgeon to the North-Eastern Hospital for Children; and Demonstrator of Anatomy in University College; 81, Wimpole street, Cavendish square.

1870 GODSON, CLEMENT, M.D., Assistant-Physician-Accoucheur to St. Bartholomew's Hospital; Consulting Physician to the City of London Lying-in Hospital; 9, Grosvenor street, Grosvenor square.

1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; Swinnerton Lodge, near Dartmouth, Devon. C. 1864-5. *Referee*, 1860-3. *Lib. Com.* 1863. *Trans.* 2.

Elected

1883 GOODHART, JAMES FREDERIC, M.D., Assistant Physician to, and Curator of the Museum at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 25, Weymouth street, Portland place.

1877 GOULD, ALFRED PEARCE, M.S., Assistant Surgeon to the Middlesex Hospital; Surgeon to the North-west London Hospital; 16, Queen Anne street, Cavendish square. *Trans.* 1.

1873 GOWERS, WILLIAM RICHARD, M.D., Assistant Professor of Clinical Medicine in University College, and Physician to University College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 50, Queen Anne street, Cavendish square. *Lib. Com.* 1884. *Trans.* 6.

1851 †GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 34, Finsbury square.

1846 GREAM, GEORGE THOMPSON, M.D., Physician-Accoucheur to H.R.H. the Princess of Wales; Mixbury, Eastbourne, Sussex. C. 1863.

1868 GREEN, T. HENRY, M.D., Physician to, and Lecturer on Pathology at, Charing Cross Hospital; Assistant-Physician to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square. *Referee*, 1882-4.

1875 *GREENFIELD, WILLIAM SMITH, M.D., Professor of General Pathology in the University of Edinburgh; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee*, 1881.

1843 †GREENHALGH, ROBERT, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children, and to the City of London Lying-in Hospital; 35, Cavendish square. C. 1871-2. *Referee*, 1876-7. *Trans.* 1.

1860 GREENHOW, EDWARD HEADLAM, M.D., F.R.S., Consulting Physician to the Middlesex Hospital; and Consulting Physician to the Western General Dispensary; Castle Lodge, Reigate. C. 1876-7. *Referee*, 1870-5. *Trans.* 3.

Elected

1882 GRESSWELL, DAN ASTLEY, M.B., 30, Great Russell street, Bloomsbury.

1868 GRIGG, WILLIAM CHAPMAN, M.D., Assistant Obstetric Physician to the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in-Hospital; Assistant-Physician to the Victoria Hospital for Children; 6, Curzon street, Mayfair.

1852 GROVE, JOHN, Fyning, Austen road, Guildford.

1860 GUENEAU DE MUSSY, HENRI, M.D.; 15, Rue du Cirque, Paris. *Lib. Com.* 1863-5.

1849 †GULL, SIR WILLIAM WITHEY, Bart., M.D., D.C.L., LL.D., F.R.S., Physician-Extraordinary to the Queen; Member of the Senate of the University of London; Consulting Physician to Guy's Hospital; 74, Brook street, Grosvenor square. C. 1864. V.P. 1874. *Referee*, 1855-63. *Trans.* 4.

1883 GUNN, ROBERT MARCUS, M.B., 108, Park street, Grosvenor square.

1854 HABERSHON, SAMUEL OSBORNE, M.D., 70, Brook street, Grosvenor square. S. 1867. C. 1869-70. V.P. 1881-2. *Referee*, 1862-6, 1868, 1871-80. *Trans.* 3.

1881 HALL, FRANCIS DE HAVILLAND, M.D., Assistant Physician, and Physician to the Throat Department, and Lecturer on Forensic Medicine at the Westminster Hospital; Physician to St. Mark's Hospital; 46, Queen Anne street, Cavendish square.

1870 HAMILTON, ROBERT, Surgeon to the Royal Southern Hospital, Liverpool; 1 Prince's road, Liverpool.

1874 HARDIE, GORDON KENMURE, M.D., Deputy Inspector General of Hospitals; Florence road, Ealing, and Duff House, Banff, N.B.

1856 †HARE, CHARLES JOHN, M.D., late Professor of Clinical Medicine in University College, London, and late Physician to University College Hospital; Berkeley House, 15, Manchester square. C. 1873-4.

Elected

1857 HARLEY, GEORGE, M.D., F.R.S. 25, Harley street, Cavendish square. C. 1871-2. *Referee*, 1865-70, 1873-6. *Sci. Com.* 1862-3. *Trans.* 1.

1864 HARLEY, JOHN, M.D., F.L.S., Physician to, and Lecturer on General Anatomy and Physiology at, St. Thomas's Hospital; 39, Brook street, Grosvenor square. S. 1875-7. C. 1879-80. *Referee*, 1871-4, 1882-4. *Sci. Com.* 1879. *Trans.* 9.

1859 HARRIS, FRANCIS, M.D., F.L.S., 24, Cavendish square.

1880 HARRIS, VINCENT DORMER, M.D., Assistant Physician to the Victoria Park Hospital; Demonstrator of Physiology at St. Bartholomew's Hospital; 39, Wimpole street, Cavendish square.

1872 HARRIS, WILLIAM H., M.D., Deputy Surgeon-General, Madras Army (retired); late Professor of Midwifery and Diseases of Women and Children, Madras Medical College.

1870 HARRISON, REGINALD, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Surgery at the School of Medicine; 38, Rodney street, Liverpool. *Trans.* 1.

1854 HAVILAND, ALFRED.

1870 HAWARD, J. WARRINGTON, Surgeon to St. George's Hospital; Surgeon to the Hospital for Sick Children; 16, Savile row, Burlington gardens. *Lib. Com.* 1881–4. *Trans.* 1.

1838 †HAWKINS, CHARLES, Inspector of Anatomical Schools in London; 27, Savile row, Burlington gardens. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Referee*, 1859-60. *Lib. Com.* 1843. *Trans.* 2.

1848 †HAWKSLEY, THOMAS, M.D., Consulting Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 62, Green street, Grosvenor square.

1875 HAYES, THOMAS CRAWFORD, M.D., Physician-Accoucheur and Physician for Diseases of Women and Children to King's College Hospital; 17, Clarges street, Piccadilly.

1860 HAYWARD, HENRY HOWARD, Surgeon Dentist to, and Lecturer on Dental Surgery at, St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878-9.

Elected

1861 HAYWARD, WILLIAM HENRY, Chapel Ash, Wolverhampton.

1848 *HEALE, JAMES NEWTON, M.D.

1865 HEATH, CHRISTOPHER, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. C. 1880. *Lib. Com.* 1870-3. *Trans.* 3.

1850 HEATON, GEORGE, M.D., Boston, U.S.

1882 HENSLEY, PHILIP J., M.D., Assistant Physician and Lecturer on Forensic Medicine to St. Bartholomew's Hospital; 4, Henrietta street, Cavendish square.

1821 HERBERSKI, VINCENT, M.D., Professor of Medicine in the University of Wilna.

1877 HERMAN, GEORGE ERNEST, M.B., Obstetric Physician to, and Lecturer on Midwifery at, the London Hospital; 7, West street, Finsbury circus. *Trans.* 1.

1877 HERON, GEORGE ALLAN, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; Assistant Physician to the West London Hospital for the Paralysed and Epileptic; 40, Margaret street, Cavendish square.

1883 HERRINGHAM, WILMOT PARKER, M.B., 22, Bedford square.

1843 HEWETT, SIR PRESCOTT GARDNER, Bart., F.R.S., Serjeant-Surgeon to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; Corresponding Member of the Academy of Medicine, and of the "Société de Chirurgie," Paris; Chesnut Lodge, Horsham, Sussex. C. 1859. V.P. 1866-7. *Referee*, 1850-8, 1860-5, 1868-83. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 7.

1855 HEWITT, W. M. GRAILY, M.D., Professor of Midwifery in University College, London, and Obstetric Physician to University College Hospital; 36, Berkeley square. C. 1876. *Referee*, 1868-75, 1877-84. *Lib. Com.* 1868, 1874.

Elected

1880 HICKS, CHARLES CYRIL, M.D., Wokingham, Berks.

1873 HIGGENS, CHARLES, Assistant Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, Guy's Hospital; 38, Brook street, Grosvenor square. *Trans.* 2.

1862 HILL, M. BERKELEY, M.B., *Secretary*, Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; Surgeon to the Lock Hospital; 66, Wimpole street, Cavendish square. C. 1878-9. S. 1881-4. *Trans.* 1.

1867 HILL, SAMUEL, M.D., 22, Mecklenburgh square.

1861 *HOFFMEISTER, SIR WILLIAM CARTER, M.D., Surgeon to H.M. the Queen in the Isle of Wight; Clifton House, Cowes, Isle of Wight.

1843 †HOLDEN, LUTHER, Consulting Surgeon to St. Bartholomew's Hospital, to the Metropolitan Dispensary, and to the Foundling Hospital; Pinetoft, Ipswich. C. 1859. L. 1865. V.P. 1874. *Referee*, 1866-7. *Lib. Com.* 1858.

1879 HOLLAND, PHILIP ALEXANDER, M.A.

1868 HOLLIS, WILLIAM AINSLIE, M.A., M.D., Assistant-Physician to the Sussex County Hospital; 8, Cambridge road, Brighton.

1861 HOLMAN, WILLIAM HENRY, M.B., 68, Adelaide road, South Hampstead.

1856 HOLMES, TIMOTHY, M.A. Surgeon to St. George's Hospital; Surgeon-in-Chief to the Metropolitan Police Force; 18, Great Cumberland place, Hyde park. C. 1869-70 L. 1873-7. S. 1878-80. V.P. 1881-2. *Referee*, 1866-8, 1872, 1883-4. *Sci. Com.* 1867. *Lib. Com.* 1863-5. *Trans.* 8.

1846 †HOLT, BARNARD WIGHT, Consulting Surgeon to the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington gardens. C. 1862-3. V.P. 1879-80.

Elected

1846 †HOLTHOUSE, CARSTEN, 35, Essex street, Strand. C. 1863. *Referee* 1870-6. *Lib Com.* 1859-60.

1878 HOOD, DONALD WILLIAM CHARLES, M.D., Assistant Physician to the West London Hospital; 43, Green street, Park lane.

1883 HORSLEY, VICTOR A., Assistant Professor of Pathological Anatomy, University College, London; Superintendent of the Brown Institution, Wandsworth road; 129, Gower street.

1878 HOUGHTON, WALTER B., M.D., Church Villa, Warrior square, St. Leonards-on-Sea.

1865 HOWARD, BENJAMIN, M.D., New York, U.S.

1865 HOWARD, EDWARD, M.D.

1881 HOWARD, HENRY, M.B., Stockwell Fever Hospital.

1874 HOWSE, HENRY GREENWAY, M.S., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; Examiner in Anatomy in the University of London; 10, St. Thomas's street, Southwark. *Sci. Com.* 1879. *Trans.* 2.

1884 HUGGARD, WILLIAM R., M,D., Sussex House, Hammersmith.

1857 HULKE, JOHN WHITAKER, F.R.S., *Librarian*, Surgeon to the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. S. 1876-7. L. 1879-84. *Sci. Com.* 1867. *Lib. Com.* 1864-8. *Trans.* 8.

1844 †HUMBY, EDWIN, M.D., 83, Hamilton terrace, St. John's wood. C. 1866-7.

1855 HUMPHRY, GEORGE MURRAY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Surgery in the University of Cambridge. *Trans.* 6.

1882 HUMPHRY, LAURENCE, M.B., 3, Trinity street, Cambridge.

1873 HUNTER, SIR W. GUYER, M.D., Hon. Surgeon to H.M. the Queen; late Principal of, and Professor of Medicine in, Grant Medical College, Bombay; Surgeon-General Bombay Army; 21, Norfolk crescent, Hyde park.

Elected

1849 HUSSEY, EDWARD LAW, Consulting Surgeon to the County Lunatic Asylum and the Warneford Asylum; 8, St. Aldate's, Oxford. *Trans.* 1.

1856 HUTCHINSON, JONATHAN, F.R.S., Consulting Surgeon to, and Emeritus Professor of Surgery at, the London Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. *Referee*, 1876-81, 1883-4. *Lib. Com.* 1864-5. *Trans.* 11. *Pro.* 2.

1820 HUTCHINSON, WILLIAM, M.D.

1840 †HUTTON, CHARLES, M.D., Consulting Physician to the General Lying-in Hospital; 26, Lowndes street, Belgrave square. C. 1858-9.

1847 IMAGE, WILLIAM EDMUND, Herringswell House, Mildenhall, Suffolk. *Trans.* 1.

1856 INGLIS, CORNELIUS, M.D., Cairo. [Athenæum Club, Pall Mall.]

1871 JACKSON, J. HUGHLINGS, M.D., F.R.S., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square.

1841 †JACKSON, PAUL, 51, Wellington road, St. John's Wood. C. 1862.

1863 JACKSON, THOMAS VINCENT, Senior Surgeon to the Wolverhampton and Staffordshire General Hospital; 47, Waterloo road, south, Wolverhampton.

1883 JACOBSON, WALTER HAMILTON ACLAND, B.A., M.B., Assistant Surgeon to Guy's Hospital; Surgeon to the Royal Hospital for Children and Women; 41, Finsbury square.

1825 JAMES, JOHN B., M.D.

1883 *JENKINS, EDWARD JOHNSTONE, M.B., The Australian Club, Sydney, New South Wales.

Elected

1851 †JENNER, SIR WILLIAM, Bart., M.D., K.C.B., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Emeritus Professor of Clinical Medicine in University College, London; and Consulting Physician to University College Hospital; Member of the Senate of the University of London; 63, Brook street, Grosvenor square. C. 1864. V.P. 1875. *Referee*, 1855, 1859-63. *Trans.* 3.

1881 JENNINGS, WILLIAM OSCAR, M.D., 8, Rue Roy, Paris.

1884 JESSETT, FREDERIC BOWREMAN, Surgeon to the Royal General Dispensary; 16, Upper Wimpole street.

1883 JESSOP, WALTER H. H., M.B., Assistant Demonstrator of Anatomy at St. Bartholomew's Hospital; 73, Harley street.

1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Génevois."

1847 †JOHNSON, GEORGE, M.D., F.R.S., *President*, Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. V.P. 1870. P. 1884. L. 1878-80. *Referee*, 1853-61, 1864-9. *Lib. Com.* 1860-1. *Trans.* 10.

1881 JOHNSON, GEORGE LINDSAY, M.A., M.D., Fern Lea, Highfield hill, Upper Norwood.

1848 JOHNSTONE, ATHOL ARCHIBALD WOOD, Consulting Surgeon to the Royal Alexandra Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Lib. Com.* 1860. *Trans.* 1.

1876 JONES, LESLIE HUDSON, M.D., Limefield House, Cheetham hill, Manchester.

1875 *JONES, PHILIP SYDNEY, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, Sydney University; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., 1, Gresham buildings, Basinghall street.]

Elected

1859 JONES, WILLIAM PRICE, M.D., Claremont road, Surbiton, Kingston.

1865 JORDAN, FURNEAUX, Surgeon to the Queen's Hospital, and Professor of Surgery at the Queen's College, Birmingham; Gate House, Edmund street, Birmingham.

1881 JULER, HENRY EDWARD, Assistant Surgeon Royal Westminster Ophthalmic Hospital; Junior Ophthalmic Surgeon to St. Mary's Hospital; 77, Wimpole street, Cavendish square.

1816 *KAUFFMANN, GEORGE HERMANN, M.D., Hanover.

1882 KEETLEY, CHARLES R. B., Senior Surgeon to the West London Hospital; Surgeon to the Surgical Aid Society; 30A, George street, Hanover square.

1972 KELLY, CHARLES, M.D., Professor of Hygiene at King's College, London, and Medical Officer of Health for the West Sussex Combined Sanitary District; Broadwater road, Worthing, Sussex.

1848 *KENDELL, DANIEL BURTON, M.D., Heath House, Wakefield, Yorkshire.

1877 *KHORY, RUSTONJEE NASERWANJEE, M.D., Physician to the Parell Dispensary, Bombay; Girgaum road, Bombay.

1857 KIALLMARK, HENRY WALTER, 5, Pembridge gardens, Bayswater.

1881 KIDD, PERCY, M.A., M.D., Assistant Physician to the Hospital for Consumption, Brompton; 60, Brook street, Grosvenor square. *Trans.* 2.

1851 †KINGDON, JOHN ABERNETHY, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.

1883 KNAPTON, GEORGE, Strathgyle, Portswood, Southampton.

1840 †LANE, SAMUEL ARMSTRONG, Consulting Surgeon to St. Mary's Hospital and to the Lock Hospital; Greenford, Middlesex. C. 1849-50. V.P. 1865. *Referee*, 1850.

Elected

1884 LANE, WILLIAM ARBUTHNOT, M.S., Assistant Surgeon to the Hospital for Sick Children; 14, St. Thomas's street, Southwark. *Trans.* 1.

1882 LANG, WILLIAM, Ophthalmic Surgeon to the Middlesex Hospital; 26, Upper Wimpole street, Cavendish square.

1865 LANGTON, JOHN, Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 2, Harley street, Cavendish square. C. 1881-2. *Lib. Com.* 1879-80.

1873 *LARCHER, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.

1862 LATHAM, PETER WALLWORK, M.A., M.D., Downing Professor of Medicine, Cambridge University; Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.

1816 LAWRENCE, G. E.

1884 LAWSON, GEORGE, Surgeon to the Royal London Ophthalmic Hospital and to the Middlesex Hospital; 12, Harley street.

1880 LAYCOCK, GEORGE LOCKWOOD, M.B., 12, Upper Berkeley street, Portman square.

1882 LEDWICH, EDWARD L'ESTRANGE, Lecturer on Surgical and Descriptive Anatomy in the Ledwich School of Medicine, Dublin; 23, Upper Leeson Street, Dublin.

1843 †LEE, HENRY, Consulting Surgeon to St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868-9. *Referee*, 1855, 1866-8. *Sci. Com.* 1867. *Trans.* 13. *Pro.* 2.

1884 LEE, ROBERT JAMES, M.D., 6, Savile row.

1883 LEESON, JOHN RUDD, M.D., C.M., 6, Clifden road, Twickenham.

Elected

1869 LEGG, JOHN WICKHAM, M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, St. Bartholomew's Hospital; 47, Green street, Park lane. *Referee*, 1882-4. *Lib. Com.* 1878-84. *Trans.* 2.

1836 LEIGHTON, FREDERICK, M.D.

1872 LIEBREICH, RICHARD, Consulting Ophthalmic Surgeon to St. Thomas's Hospital; Paris.

1878 LISTER, SIR JOSEPH, Bart., D.C.L., LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen; Professor of Clinical Surgery at King's College, London; and Surgeon to King's College Hospital; 12, Park crescent, Regent's park.

1872 *LITTLE, DAVID, M.D., Senior Surgeon to the Royal Eye Hospital, Manchester; 21, St. John street, Manchester.

1871 LITTLE, LOUIS STROMEYER, Shanghai, China.

1870 LIVINGSTON, JOHN, M.D., New Barnet, Hertfordshire.

1819 LLOYD, ROBERT, M.D.

1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.

1881 LOCKWOOD, CHARLES BARRETT, Surgeon to the Great Northern Hospital, and Demonstrator of Anatomy at St. Bartholomew's Hospital; 8, Serjeants' Inn, Fleet street.

1860 LONGMORE, THOMAS, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff, and Professor of Military Surgery, Army Medical School, Netley, Southampton; Woolston Lawn, Woolston, Hants. *Trans.* 2.

1836 LÖWENFELD, JOSEPH S., M.D., Berbice.

1871 LOWNDS, THOMAS MACKFORD, M.D., late Professor of Anatomy and Physiology at Grant Medical College, Bombay; Egham Hill, Surrey.

Elected

1877 LOWNE, BENJAMIN THOMPSON, Lecturer on Physiology, Middlesex Hospital Medical School; 65, Cambridge gardens, Notting hill. *Referee*, 1884.

1881 LUCAS, RICHARD CLEMENT, Senior Assistant Surgeon to, and Demonstrator of Operative and Practical Surgery at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 18, Finsbury square.

1883 LUND, EDWARD, Professor of Surgery, Owens College; Consulting Surgeon to the Manchester Royal Infirmary; 22, St. John street, Manchester.

1857 LYON, FELIX WILLIAM, M.D., 5, North Charlotte street, Edinburgh.

1882 LYONS, ISIDORE, Dental Surgeon to the Evelina Hospital for Children; Assistant Dental Surgeon to St. Bartholomew's Hospital.

1867 MABERLY, GEORGE FREDERICK, 7, Manor terrace, Brixham, South Devon.

1873 MACCARTHY, JEREMIAH, M.A., Surgeon to, and Lecturer on Physiology at, the London Hospital; 15, Finsbury square. *Lib. Com.* 1882-4.

1867 MACCORMAC, SIR WILLIAM, M.A., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; Examiner in Surgery at the University of London; 13, Harley street. C. 1884. *Trans.* 1.

1862 *M'DONNELL, ROBERT, M.D., F.R.S., Surgeon to Steevens' Hospital; 89, Merrion square west, Dublin. *Trans.* 2.

1880 *MACFARLANE, ALEXANDER WILLIAM, M.D., Consulting Physician to the Kilmarnock Fever Hospital and Infirmary; Walmer, Kilmarnock, N.B.

1866 MACGOWAN, ALEXANDER THORBURN, Vyvyan House, Clifton, near Bristol.

1880 MCHARDY, MALCOLM MACDONALD, Ophthalmic Surgeon to King's College Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 5, Savile row.

Elected

1822 MACINTOSH, RICHARD, M.D.

1859 *M'INTYRE, JOHN, M.D., Odiham, Hants.

1873 MACKELLAR, ALEXANDER OBERLIN, M.S.I., Assistant Surgeon to St. Thomas's Hospital; 22, George street, Hanover square.

1881 MACKENZIE, STEPHEN, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; Physician to the Royal London Ophthalmic Hospital; 26, Finsbury square. *Trans.* 1.

1876 MACKEY, EDWARD, M.D., 1, Brunswick road, Hove, Brighton.

1854 *MACKINDER, DRAPER, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

1879 MACLAGAN, THOMAS JOHN, M.D., Physician-in-Ordinary to their R.H. the Prince and Princess Christian of Schleswig-Holstein; 9, Cadogan place, Belgrave square.

1860 MACLEAN, JOHN, M.D., 24, Portman street, Portman square.

1876 MACNAMARA, CHARLES, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon-Major Bengal Medical Service; Fellow of the Calcutta University; 13, Grosvenor street. *Referee*, 1884.

1881 MACREADY, JONATHAN F. C. H., Surgical Registrar to St. Bartholomew's Hospital; Surgeon to the Great Northern Hospital; 125, Harley street, Cavendish square.

1880 MADDICK, EDMUND DISTIN, 17, Upper Wimpole street.

1880 MAKINS, GEORGE HENRY, St. Thomas's Hospital, Albert Embankment.

1876 MALLAM, BENJAMIN, Meadow Side, Leacroft road, Staines.

1855 MARCET, WILLIAM, M.D., F.R.S.; 39, Grosvenor street. C. 1871. *Referee*, 1866-70, 1883-4. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.

Elected

1867 MARSH, F. HOWARD, Assistant Surgeon to St. Bartholomew's Hospital; Surgeon to the Hospital for Sick Children, Great Ormond street; 36, Bruton street, Berkeley square. C. 1882-3. *Lib. Com.* 1880-1. *Trans.* 3.

1838 MARSH, THOMAS PARR, M.D.

1851 †MARSHALL, JOHN, F.R.S., Professor of Anatomy to the Royal Academy of Arts; Professor of Surgery in University College, London, and Surgeon to University College Hospital; 10, Savile row, Burlington gardens. C. 1866. V.P. 1875-6. P. 1882-4. *Referee*, 1867, 1871-4, 1877-81. *Trans.* 3.

1884 MARTIN, SIDNEY HARRIS COX, M.B., 105, Haverstock hill.

1864 MASON, FRANCIS, Surgeon to, and Lecturer on Operative Surgery at, St. Thomas's Hospital; 5, Brook street, Grosvenor square. C. 1880-81. *Trans.* 1.

1883 MAUDSLEY, HENRY, M.D., University College Hospital, Gower street.

1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1870 MEADOWS, ALFRED, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 27, George street, Hanover square. *Lib. Com.* 1875-7.

1865 MEDWIN, AARON GEORGE, M.D., Dental Surgeon to the Royal Kent Dispensary, 34, Bruton street, Berkeley square.

1880 MEREDITH, WILLIAM APPLETON, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 6, Queen Anne street, Cavendish square.

1874 MERRIMAN, JOHN J., 45, Kensington square.

1815 MEYER, AUGUSTUS, M.D., St. Petersburg.

1840 MIDDLEMORE, RICHARD, Consulting Surgeon to th Birmingham Eye Hospital; The Limes, Bristol road, Edgbaston, Birmingham.

1854 MIDDLESHIP, EDWARD ARCHIBALD.

Elected

1882 MILLS, JOSEPH, 15, Henrietta street, Cavendish square.

1873 MILNER, EDWARD, Surgeon for Out-Patients to the Lock Hospital ; 32, New Cavendish street, Portland place.

1883 MONEY, ANGEL, M.D., Assistant Physician to the City of London Hospital for Diseases of the Chest, Victoria park ; 14, Langham place, Regent street. *Trans.* 1.

1873 MOORE, NORMAN, M.D., Assistant Physician and Warden of the College and Lecturer on Comparative Anatomy, Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital ; the College, St. Bartholomew's Hospital.

1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. C. 1880-1. *Lib. Com.* 1862-3. *Trans.* 1.

1861 MORGAN, JOHN EDWARD, M.D., Physician to the Manchester Royal Infirmary, and Professor of Medicine in the Owens College, Manchester ; 1, St. Peter's square, Manchester.

1878 MORGAN, JOHN HAMMOND, M.A., Assistant Surgeon to the Charing Cross Hospital, and to the Hospital for Sick Children, Great Ormond street ; 68, Grosvenor street. *Trans.* 1.

1874 MORRIS, HENRY, M.A., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 2, Mansfield street, Portland place. *Referee*, 1882-4. *Trans.* 7.

1879 MORRIS, MALCOLM ALEXANDER, Lecturer on Skin Diseases at St. Mary's Hospital ; 63, Montagu square.

1851 MOUAT, FREDERIC JOHN, M.D., Deputy Inspector-General of Hospitals ; Medical Inspector to the Local Government Board ; and Member of the Senate of the University of Calcutta.

1868 MOXON, WALTER, M.D., F.L.S., Physician to, and Lecturer on Medicine at, Guy's Hospital ; 6, Finsbury Circus. *Referee*, 1879-84. *Trans.* 1.

1879 MUNK, WILLIAM, M.D., Harveian Librarian, Royal College of Physicians ; Consulting Physician to the Royal Hospital for Incurables ; 40, Finsbury square.

Elected

1873 MURRAY, IVOR, M.D., F.R.S. Ed. 8, Huntriss Row, Scarborough.

1880 MURRELL, WILLIAM, M.D., Assistant Physician to the Royal Hospital for Diseases of the Chest; Assistant Physician to, and Lecturer on Materia Medica and Therapeutics at, the Westminster Hospital; 38, Weymouth street, Portland place. *Trans.* 1.

1863 MYERS, ARTHUR BOWEN RICHARDS, Surgeon to the 1st Battalion, Coldstream Guards; Hospital, Vincent square, Westminster. C. 1878-9. *Lib. Com.* 1877.

1882 MYERS, A. T., M.D., Medical Registrar, St. George's Hospital; 24, Clarges street, Piccadilly.

1881 NALL, SAMUEL, M.B., Disley, Cheshire.

1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine in the University of Melbourne; 166, Collins street east, Melbourne, Victoria.

1835 †NELSON, THOMAS ANDREW, M.D., 10, Nottingham terrace, York gate, Regent's park. *Lib. Com.* 1841.

1877 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital; Ophthalmic Surgeon to the Hospital for Sick Children; 5, Wimpole street, Cavendish square.

1843 †NEWTON, EDWARD, 85, Gloucester terrace, Hyde Park. C. 1863-4.

1868 NICHOLLS, JAMES, M.D., 68, Duke street, Chelmsford, Essex.

1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants. *Lib. Com.* 1857.

1847 *NOURSE, WILLIAM EDWARD CHARLES, Bouverie House, Mount Radford, Exeter.

1864 NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1870 NUNNELEY, FREDERICK BARHAM, M.D. *Trans.* 2.

Elected

1880 O'CONNOR, BERNARD, A.B., M.D., Physician to the North London Hospital for Consumption, and Physician to the Westminster General Dispensary; 6, Nottingham terrace, York Gate, Regent's Park.

1847 O'CONNOR, THOMAS, March, Cambridgeshire.

1880 OGILVIE, GEORGE, M.B., Lecturer on Experimental Physics at the Westminster Hospital; 13, Welbeck street, Cavendish square.

1880 OGILVIE, LESLIE, M.B., Lecturer on Comparative Anatomy at the Westminster Hospital; 46, Welbeck street, Cavendish square.

1858 OGLE, JOHN WILLIAM, M.D., Consulting Physician to St. George's Hospital; 30, Cavendish square. C. 1873. *Referee*, 1864-72. *Trans.* 4.

1855 *OGLE, WILLIAM, M.A., M.D., Physician to the Derby Infirmary; The Elms, Duffield road, Derby.

1860 OGLE, WILLIAM, M.D., Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. *Lib. Com.* 1871-5. *Trans.* 4.

1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].

1883 *OLIVER, THOMAS, M.D., Lecturer on Practical Physiology, University of Durham; and Physician to the Newcastle-upon-Tyne Infirmary; 3, Eldon square, Newcastle-upon-Tyne.

1871 *O'NEILL, WILLIAM, M.D., Physician to the Lincoln Lunatic Hospital, Silver street, Lincoln.

1873 ORD, WILLIAM MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; Examiner in Medicine at the University of London; 7, Brook street, Hanover square. *Referee*, 1884. *Trans.* 6.

1877 ORMEROD, JOSEPH ARDERNE, M.D., Assistant Demonstrator of Physiology to St. Bartholomew's Hospital; Assistant Physician to the National Hospital for the Paralysed and Epileptic, Queen square, and to the City of London Hospital for Diseases of the Chest, Victoria Park; 25, Upper Wimpole street.

Elected

1875 OSBORN, SAMUEL, 10, Maddox street, Regent street; and Datchet, Bucks.

1879 OWEN, EDMUND, Surgeon to St. Mary's Hospital; Surgeon to the Hospital for Sick Children; 49, Seymour street, Portman square. *Trans.* 1.

1882 OWEN, HERBERT ISAMBARD, M.D., Assistant Physician to, and Lecturer on Materia Medica at, St. George's Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 5, Hertford street, May Fair.

1874 PAGE, HERBERT WILLIAM, M.A., M.C., Surgeon (with charge of out-patients) to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 146, Harley street, Cavendish square. *Referee*, 1884. *Trans.* 2.

1847 *PAGE, WILLIAM BOUSFIELD, Consulting Surgeon to the Cumberland Infirmary, 78, Carlisle street, Carlisle. *Trans.* 2.

1840 †PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Vice-Chancellor of the University of London; 1, Harewood place, Hanover square. C. 1848-9. V.P. 1861. T. 1867. P. 1875-6. *Referee*, 1844-6, 1848, 1851-60, 1862-6, 1868-74. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 12.

1858 *PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; Ripon, Yorkshire.

1847 PARKER, NICHOLAS, M.D., Paris.

1873 PARKER, ROBERT WILLIAM, Surgeon to the East London Hospital for Children; 8, Old Cavendish street. *Trans.* 3.

1841 PARKIN, JOHN, M.D., 5, Codrington place, Brighton.

1883 PASTEUR, WILLIAM, M.B., 19, Queen street, May Fair.

1865 PAVY, FREDERICK WILLIAM, M.D., F.R.S., Physician to Guy's Hospital; 35, Grosvenor street. C. 1883-4. *Referee*, 1871-82. *Trans.* 1.

1869 PAYNE, JOSEPH FRANK, M.D., Assistant-Physician to, and Lecturer on Pathological Anatomy at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. *Sci. Com.* 1879. *Lib. Com.* 1878-84.

Elected

1879 PEEL, ROBERT, 120, Collins street east, Melbourne, Victoria.

1856 PEIRCE, RICHARD KING, Woodside, Windsor forest, Berks.

1830 PELECHIN, CHARLES P., M.D., St. Petersburg.

1855 *PEMBERTON, OLIVER, Senior Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham; 12, Temple row, Birmingham. *Trans.* 1.

1874 PENHALL, JOHN THOMAS, 5, Eversfield place, St. Leonard's, Sussex.

1870 PERRIN, JOHN BESWICK, Vernon House, Leigh, Lancashire.

1879 *PESIKAKA, HORMASJI DOSABHAI, Marine Lines, Bombay.

1878 *PHILIPSON, GEORGE HARE, M.D., M.A., D.C.L., Professor of Medicine at Durham University; Senior Physician to the Newcastle-upon-Tyne Infirmary; 7, Eldon square, Newcastle-upon-Tyne.

1883 PHILLIPS, CHARLES DOUGLAS F., M.D., 10, Henrietta street, Cavendish square, W.

1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 18, Portman street, Portman square. C. 1884. *Referee*, 1882-3. *Sci. Com.* 1870. *Lib. Com.* 1879-81.

1841 †PITMAN, SIR HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital, and to the Royal General Dispensary, St. Pancras; 28, Gordon square. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1. *Referee*, 1849-50. *Lib. Com.* 1847.

1884 PITT, GEORGE NEWTON, M.D., Assistant Physician to the Shadwell Hospital for Children; 84, Ashburn place, Kensington.

1871 POLLOCK, ARTHUR JULIUS, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, Charing Cross Hospital; Physician to the Foundling Hospital; 85, Harley street, Cavendish square.

Elected

1845 †POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1. *Referee*, 1858, 1864-9, 1877-84. *Trans.* 4.

1865 POLLOCK, JAMES EDWARD, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. C. 1882-3. *Referee*, 1872-81.

1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence in University College, London; Assistant-Physician to University College Hospital; Physician to the Royal Infirmary for Children and Women, Waterloo road; Examiner in Forensic Medicine at the University of London; 30, Wimpole street. *Trans.* 1.

1846 POTTER, JEPHSON, M.D., F.L.S.

1842 POWELL, JAMES, M.D.

1867 POWELL, RICHARD DOUGLAS, M.D., *Secretary*, Physician to the Middlesex Hospital; Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 62, Wimpole street, Cavendish sq. S. (Oct.) 1883-4. *Referee*, 1879-83. *Trans.* 2.

1867 POWER, HENRY, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; 37A, Great Cumberland place, Hyde park. C. 1882-3. *Referee*, 1870-81. *Sci. Com.* 1870. *Lib. Com.* 1872-8.

1857 PRIESTLEY, WILLIAM OVEREND, M.D., *Vice-President*, Consulting Physician-Accoucheur to King's College Hospital, and to the St. Marylebone Infirmary; 17, Hertford street, Mayfair. C. 1874-5. V.P. 1884. *Referee*, 1867-73, 1877-83. *Sci. Com.* 1863.

1883 PRINGLE, JOHN JAMES, M.B., C.M., 35, Bruton Street, Berkeley square.

1874 PURVES, WILLIAM LAIDLAW, Aural Surgeon to Guy's Hospital; 20, Stratford place, Oxford street. *Trans.* 2.

Elected

1878 PYE, WALTER, Surgeon (with charge of out-patients) to St. Mary's Hospital; 4, Sackville street, Piccadilly.

1877 PYE-SMITH, PHILIP HENRY, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital; Examiner in Physiology at the University of London; 54, Harley street, Cavendish square.

1850 †QUAIN, RICHARD, M.D., F.R.S., Consulting Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. V.P. 1878-9. *Sci. Com.* 1863. *Trans.* 1.

1835 †QUAIN, RICHARD, F.R.S., Surgeon-Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery, University College, London, and Consulting Surgeon to University College Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Referee*, 1845-6, 1848, 1858-9. *Lib. Com.* 1846. *Trans.* 1. *Pro.* 2.

1852 †RADCLIFFE, CHARLES BLAND, M.D., *Treasurer*, Consulting Physician to the Westminster Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square. C. 1867-8. V.P. 1879-80. T. 1881-4. *Referee*, 1862-6, 1870-8.

1871 RALFE, CHARLES HENRY, M.D., M.A., Assistant Physician to the London Hospital, and late Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square.

1857 RANKE, HENRY, M.D., 3, Sophienstrasse, Munich.

1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Physician to the Nottingham General Hospital, Nottingham.

1869 READ, THOMAS LAURENCE, 11, Petersham terrace, Queen's gate.

1858 REED, FREDERICK GEORGE, M.D., 46, Hertford street, Mayfair. *Trans.* 1.

1821 REEDER, HENRY, M.D., Varick, Seneca County, New York, United States.

Elected

1857 REES, GEORGE OWEN, M.D., F.R.S., Consulting Physician to Guy's Hospital; 26, Albemarle street, Piccadilly. C. 1873. *Referee*, 1860-72, 1875-81. *Trans.* 1.

1882 REID, JAMES, M.D., Resident Physician to H.M. the Queen, Windsor Castle.

1884 REID, THOMAS WHITEHEAD, Surgeon to the Kent and Canterbury Hospital; 34, St. George's place, Canterbury.

1855 REYNOLDS, JOHN RUSSELL, M.D., F.R.S., *Vice-President*, Physician-in-Ordinary to H.M.'s Household; Consulting-Physician to University College Hospital; 38, Grosvenor street. C. 1870. V.P. 1883. *Referee*, 1867-9.

1865 RHODES, GEORGE WINTER, Surgeon to the Huddersfield Infirmary; Queen street south, Huddersfield.

1881 RICE, GEORGE, M.B., C.M.

1852 RICHARDSON, CHRISTOPHER THOMAS, M.B., 13, Nelson crescent, Ramsgate.

1845 †RIDGE, BENJAMIN, M.D., 8, Mount street, Grosvenor square.

1863 RINGER, SYDNEY, M.D., Professor of the Principles and Practice of Medicine in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square. C. 1881-2. *Referee*, 1873-80. *Trans.* 6.

1871 RIVINGTON, WALTER, M.S., Surgeon to, and Lecturer on Surgery at, the London Hospital; 22, Finsbury square. *Trans.* 2.

1871 *ROBERTS, DAVID LLOYD, M.D., Physician to St. Mary's Hospital, Manchester; 11, St. John street, Deansgate, Manchester.

1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Materia Medica and Therapeutics in University College, London; and Physician to University College Hospital; Physician to the Hospital for Consumption, Brompton; Examiner in Materia Medica at the University of London; 53, Harley street, Cavendish square.

Elected

1857 ROBERTSON, JOHN CHARLES GEORGE, Medical Superintendent of the Cavan District Lunatic Asylum; Monaghan, Ireland.

1873 ROBERTSON, WILLIAM H., M.D., Consulting Physician to the Devonshire Hospital and Buxton Bath Charity; Buxton, Derbyshire.

1843 RODEN, WILLIAM, M.D., Morningside Lodge, Kidderminster.

1850 ROPER, GEORGE, M.D., Consulting Physician to the Eastern Division of the Royal Maternity Charity; Physician to the Royal Infirmary for Children and Women, Waterloo Bridge road [19, Ovington gardens, S.W.]. C. 1879-80.

1857 ROSE, HENRY COOPER, M.D., F.L.S., Surgeon to the Hampstead Dispensary; Penrose House, Hampstead. *Trans.* 1.

1883 ROSE, WILLIAM, M.B., Assistant Surgeon to King's College Hospital; 50, Harley street, Cavendish square.

1882 ROUTH, AMAND J. McC., M.D., B.S., Physician to the Samaritan Free Hospital for Women; Assistant Physician Accoucheur to the Charing Cross Hospital; Obstetric Physician to the St. Marylebone General Dispensary; 6, Upper Montagu street, Montagu square.

1849 †ROUTH, CHARLES HENRY FELIX, M.D., Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Lib. Com.* 1854-5. *Trans.* 1.

1863 ROWE, THOMAS SMITH, M.D., Surgeon to the Royal Sea-Bathing Infirmary; Cecil street, Margate, Kent.

1882 ROY, CHARLES SMART, M.D., F.R.S., Professor of Pathology in the University of Cambridge.

1845 RUSSELL, JAMES, M.D., Physician to the Birmingham General Hospital; 22, New Hall street, Birmingham.

1871 RUTHERFORD, WILLIAM, M.D., F.R.S., Professor of Physiology in the University of Edinburgh; 14, Douglas crescent, Edinburgh.

1856 SALTER, S. JAMES A., M.B., F.R.S., F.L.S., Basingfield, near Basingstoke, Hants. C. 1871. *Lib. Com.* 1878. *Trans.* 2.

Elected

1849 †SANDERSON, HUGH JAMES, M.D., 26, Upper Berkeley street, Portman square. C. 1872-3. *Lib. Com.* 1862-3.

1855 SANDERSON, JOHN BURDON, M.D., LL.D., F.R.S., Waynflete Professor of Physiology in the University of Oxford; 50, Banbury road, Oxford. C. 1869-70. V.P. 1882. *Referee*, 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.

1867 SANDFORD, FOLLIOTT JAMES, M.D., Market Drayton, Shropshire.

1879 SANGSTER, ALFRED, B.A., M.B., Lecturer on Skin Diseases at the Charing Cross Hospital; 6, Savile row. *Trans.* 1.

1847 †SANKEY, WILLIAM HENRY OCTAVIUS, M.D., Boreatton park, Baschurch, Shrewsbury.

1869 SANSOM, ARTHUR ERNEST, M.D., Physician (with charge of out-patients) to the London Hospital; 84, Harley street, Cavendish square. *Trans.* 2.

1845 †SAUNDERS, SIR EDWIN, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince of Wales; 13A, George street, Hanover square. C. 1872-3.

1834 SAUVAN, LUDWIG V., M.D., Warsaw.

1879 SAVAGE, GEORGE HENRY, M.D., Bethlem Royal Hospital, St. George's road, Southwark.

1859 SAVORY, WILLIAM SCOVELL, F.R.S., *Vice-President*, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to Christ's Hospital; 66, Brook street, Grosvenor square. C. 1871-2. L. 1878. V.P. 1883-4. *Referee*, 1865-70, 1873-77, 1879-82. *Sci. Com.* 1862, 1867, 1870. *Lib. Com.* 1866-8. *Trans.* 6.

1883 SCHÄFER, EDWARD ALBERT, F.R.S., Jodrell Professor of Physiology, University College, London; University College, Gower street.

1873 SCOTT, JOHN MOORE JOHNSTON, M.D., Lurgan, County Armagh.

1861 *SCOTT, WILLIAM, M.D., Senior Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.

Elected

1882 SCRIVEN, JOHN BARCLAY, Brigade Surgeon, Bengal (retired), late Professor of Anatomy, Surgery, and Ophthalmic Surgery at the Lahore Medical School; 95, Oxford gardens, Notting hill.

1863 SEDGWICK, WILLIAM, 12, Park place, Upper Baker street. C. 1884. *Trans.* 2.

1877 SEMON, FELIX, M.D., Assistant Physician for Diseases of the Throat to St. Thomas's Hospital; 59, Welbeck street, Cavendish square. *Trans.* 1.

1875 SEMPLE, ROBERT HUNTER, M.D., Physician to the Bloomsbury Dispensary; 8, Torrington square. *Sci. Com.* 1879.

1873 *SHAPTER, LEWIS, B.A., M.B., Physician to the Devon and Exeter Hospital; the Barnfield, Exeter.

1882 SHARKEY, SEYMOUR J., M.B., Assistant Physician to St. Thomas's Hospital; 2, Portland place. *Trans.* 2.

1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

1836 †SHAW, ALEXANDER, Consulting Surgeon to the Middlesex Hospital; 136, Abbey road, Kilburn. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Referee*, 1842-3, 1846-50, 1855-7, 1865. *Lib. Com.* 1843. *Trans.* 4.

1884 SHEILD, ARTHUR MARMADUKE, M.B., B.S., House Surgeon, St. George's Hospital; 83, Park street, Grosvenor square.

1859 SIBLEY, SEPTIMUS WILLIAM, 7, Harley street, Cavendish square. C. 1882-3. *Sci. Com.* 1863. *Trans.* 4.

1848 †SIEVEKING, EDWARD HENRY, M.D., Physician-Extraordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Physician to St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. *Referee*, 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.

1842 †SIMON, JOHN, C.B., D.C.L., LL.D., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee* 1851-3, 1866-81. *Trans.* 1.

Elected

1857 SIORDET, JAMES LEWIS, M.B., Villa Preti, Mentone, Alpes Maritimes, France.

1882 SMITH, CHARLES JOHN, 54, Old Steyne, Brighton.

1879 SMITH, E. NOBLE, Senior Surgeon and Surgeon to the Orthopædic Department of the Farringdon Dispensary; Orthopædic Surgeon to the British Home for Incurables; 24, Queen Anne street, Cavendish square.

1881 SMITH, EUSTACE, M.D., Physician to H.M. the King of the Belgians; Physician to the East London Children's Hospital, and to the Victoria Park Hospital for Diseases of the Chest; 5, George street, Hanover square.

1872 SMITH, T. GILBART, M.A., M.D., Assistant-Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 68, Harley street, Cavendish square. *Trans.* 1.

1866 SMITH, HEYWOOD, M.A. M.D., Physician to the Hospital for Women; Physician to the British Lying-in Hospital; 18, Harley street, Cavendish square.

1838 †SMITH, SPENCER, Consulting Surgeon to St. Mary's Hospital; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865. *Referee*, 1851-3, 1862-4, 1866-78. *Lib. Com.* 1847.

1863 SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-6. *Referee*, 1873-4, 1880-4. *Sci. Com.* 1867. *Trans.* 3.

1847 SMITH, WILLIAM J., M.D., Consulting Physician to the Weymouth Infirmary; Greenhill, Weymouth, Dorsetshire.

1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital, Greenwich.

1874 *SMITH, WILLIAM ROBERT, M.D., F.R.S. Ed., Physician to the Dispensary, Cheltenham; Bayshill Villa, Cheltenham.

1868 SOLLY, SAMUEL EDWIN, Colorado Springs, Colorado, U.S.

Elected

1865 SOUTHEY, REGINALD, M.D., Commissioner in Lunacy; 32, Grosvenor road, Westminster. C. 1881-2. S. 1883. *Referee*, 1873-80. *Trans.* 1.

1844 SPACKMAN, FREDERICK R., M.D., Harpenden, St. Alban's.

1875 SPITTA, EDMUND J., Ivy House, Clapham Common, Surrey.

1851 †SPITTA, ROBERT JOHN, M.D., East Side, Clapham Common, Surrey. C. 1878-9. *Trans.* 1.

1882 STEAVENSON, WILLIAM EDWARD, M.B., 15, Henrietta street, Cavendish square.

1854 STEVENS, HENRY, M.D., Inspector, Medical Department, Local Government Board

1859 STEWART, WILLIAM EDWARD, 16, Harley street, Cavendish square.

1879 *STIRLING, EDWARD CHARLES, late Assistant Surgeon and Lecturer on Physiology at St. George's Hospital; Adelaide, South Australia [care of T. Gemmell, Esq., 32, The Grove, Boltons, S.W.].

1856 STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham.

1865 STOKES, WILLIAM, M.D., Surgeon to the Richmond Surgical Hospital; 5, Merrion square north, Dublin. *Trans.* 1.

1884 STONHAM, CHARLES, Senior Demonstrator of Anatomy at University College, London; 129, Gower street.

1843 STORKS, ROBERT REEVE, Paris.

1858 †STREATFEILD, JOHN FREMLYN, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Professor of Clinical Ophthalmic Surgery in University College, and Senior Ophthalmic Surgeon to University College Hospital; 15, Upper Brook street, Grosvenor square. C. 1874-5. *Lib. Com.* 1867-8.

1871 STRONG, HENRY JOHN, M.D., Whitgift House, George street, Croydon.

1863 STURGES, OCTAVIUS, M.D., Physician to, and Lecturer on Medicine at, the Westminster Hospital; Assistant-Physician to the Hospital for Sick Children; 85, Wimpole street, Cavendish square. C. 1878-9. *Referee*, 1882-4.

Elected

1871 †SUTHERLAND, HENRY, M.D., Lecturer on Insanity at the Westminster Hospital; 6, Richmond terrace, Whitehall.

1860 SUTRO, SIGISMUND, M.D., Senior Physician to the German Hospital; 37A, Finsbury square.

1871 SUTTON, HENRY GAWEN, M.B., Physician to, and Lecturer on Pathology at, the London Hospital, and Physician to the City of London Hospital for Diseases of the Chest; 9, Finsbury square. *Trans.* 1.

1883 SUTTON, JOHN BLAND, Lecturer on Comparative Anatomy and Senior Demonstrator of Anatomy, Middlesex Hospital Medical College. *Trans.* 1.

1855 SUTTON, JOHN MAULE, M.D., Medical Officer of Health, Oldham; Higher Broughton, Manchester.

1861 *SWEETING, GEORGE BACON, King's Lynn, Norfolk.

1878 *SYMPSON, THOMAS, Surgeon to the Lincoln County Hospital; 3, James street, Lincoln.

1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, The Crescent, Birmingham. *Trans.* 4.

1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.

1875 TAY, WAREN, Surgeon to the London Hospital and Surgeon to the North Eastern Hospital for Children and the Hospital for Skin Diseases, Blackfriars; 4, Finsbury square.

1873 TAYLOR, FREDERICK, M.D., Assistant-Physician to, and Lecturer on Materia Medica at, Guy's Hospital; Physician to the Evelina Hospital for Sick Children; 11, St. Thomas's street, Southwark. *Trans.* 1.

1845 †TAYLOR, THOMAS, Warwick House, 1, Warwick place, Grove End road, St. John's wood.

1859 TEGART, EDWARD, 49, Jermyn street, St. James's.

1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. *Trans.* 9.

1862 THOMPSON, EDMUND SYMES, M.D., Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 33, Cavendish square. S. 1871-4. C. 1878-9. *Referee*, 1876-7. *Trans.* 1.

Elected

1857 THOMPSON, HENRY, M.D., Consulting Physician to the Middlesex Hospital; 53, Queen Anne street, Cavendish square.

1852 †THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Professor of Surgery and Pathology to the Royal College of Surgeons; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 7.

1862 THOMPSON, REGINALD EDWARD, M.D., *Vice-President*, Physician to the Hospital for Consumption, Brompton; 47, Park street, Grosvenor square. C. 1879. S. 1880-82. V.P. 1883-4. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.

1881 THOMSON, WILLIAM SINCLAIR, M.D., 40, Ladbroke grove, Kensington park gardens.

1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 22, Portman street, Portman square. *Trans.* 2.

1883 THURSFIELD, THOMAS WILLIAM, M.D., 26, The Parade, Leamington.

1875 TIBBITS, HERBERT, M.D., 68, Wimpole street.

1848 †TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 27, Seymour street, Portman square. *Referee*, 1874-81.

1880 TIVY, WILLIAM JAMES, 8, Lansdowne place, Clifton, Bristol.

1872 TOMES, CHARLES S., B.A., F.R.S., Lecturer on Anatomy and Physiology at the Dental Hospital; 37, Cavendish square. *Lib. Com.* 1879.

1867 TONGE, MORRIS, M.D., Harrow-on-the-Hill, Middlesex.

1882 TOOTH, HOWARD HENRY, M.B., 34, Harley street, Cavendish square.

1871 *TREND, THEOPHILUS W., M.D., Raeberry Lodge, Southampton.

Elected

1879 TREVES, FREDERICK, Surgeon to, and Lecturer on Anatomy at, the London Hospital; 18, Gordon square. *Trans.* 2.

1881 *TREVES, WILLIAM KNIGHT, Surgeon to the Royal Sea Bathing Infirmary for Scrofula; 31, Dalby square, Cliftonville, Margate.

1867 TROTTER, JOHN WILLIAM, late Surgeon-Major, Coldstream Guards; 4, St. Peter's terrace, York.

1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1864 TUFNELL, THOMAS JOLLIFFE, Consulting Surgeon to the City of Dublin Hospital; 58, Lower Mount street, Merrion square, Dublin. *Trans.* 1.

1862 TUKE, THOMAS HARRINGTON, M.D., Manor House, Chiswick, and 37, Albemarle street, Piccadilly.

1875 TURNER, FRANCIS CHARLEWOOD, M.A., M.D., Physician to the London Hospital; 15, Finsbury square.

1873 TURNER, GEORGE BROWN, M.D., San Remo, Italy.

1882 TURNER, GEORGE ROBERTSON, Visiting Surgeon to the Seamen's Hospital, Greenwich; Joint Lecturer on Practical Surgery at St. George's Hospital; 49, Green street, Park lane.

1881 TYSON, WILLIAM JOSEPH, M.D., Medical Officer of the Folkestone Infirmary; 10, Langhorne gardens, Folkestone.

1876 VENN, ALBERT JOHN, M.D., Obstetric Physician to the Metropolitan Free Hospital; Physician to the Victoria Hospital for Children; 8, Upper Brook street, Grosvenor square.

1870 VENNING, EDGCOMBE, 30, Cadogan place.

1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 14, Clarges street, Piccadilly.

1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy and to the French Hospital, Lisle street, Leicester square; 141, Regent street.

Elected

1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.

1854 WADDINGTON, EDWARD, Hamilton, Auckland, New Zealand.

1870 WADHAM, WILLIAM, M.D., Physician to St. George's Hospital; 14, Park lane.

1864 WAITE, CHARLES DERBY, M.B., Senior Physician to the Westminster General Dispensary; 3, Old Burlington street.

1884 WAKLEY, Thomas, Jun., 96, Redcliffe gardens.

1868 *WALKER, ROBERT, Surgeon to the Carlisle Dispensary; 2, Portland square, Carlisle.

1883 WALLER, AUGUSTUS, M.D., 26, Gordon square.

1867 *WALLIS, GEORGE, Surgeon to Addenbrooke's Hospital, Corpus Buildings, Cambridge.

1873 WALSHAM, WILLIAM JOHNSON, C.M., Assistant Surgeon to, and Demonstrator of Practical and Orthopædic Surgery at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital; 27, Weymouth street, Portland place. *Lib. Com.* 1882-4. *Trans.* 3.

1852 †WALSHE, WALTER HAYLE, M.D., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption; 41, Hyde park square. C. 1872. *Trans.* 1.

1883 *WALTERS, JAMES HOPKINS, 43, Castle street, Reading.

1851 †WALTON, HAYNES, Senior Surgeon to St. Mary's Hospital, 1, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1852 WANE, DANIEL, M.D.

1821 WARD, WILLIAM TILLEARD, Tilleards, Stanhope, Canada.

1858 WARDELL, JOHN RICHARD, M.D., Calverley park, Tunbridge Wells.

1846 WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.

1818 WARE, JOHN, Clifton Down, Bristol.

Elected

1866 WARING, EDWARD JOHN, C.I.E., M.D., 49, Clifton gardens, Maida vale. *Referee*, 1881-4.

1877 WARNER, FRANCIS, M.D., Assistant Physician to the London Hospital and to the East London Hospital for Children; Lecturer on Botany at the London Hospital; 24, Harley street, Cavendish square. *Trans.* 1.

1861 WATERS, A. T. HOUGHTON, M.D., Physician to the Royal Infirmary, and Lecturer on the Principles and Practice of Medicine, in the Liverpool Royal Infirmary School of Medicine; 69, Bedford street, Liverpool. *Trans.* 3.

1879 WATERS, JOHN HENRY, M.D., C.M., 101, Jermyn street.

1878 WATNEY, HERBERT, M.D., 1, Wilton crescent, Belgrave square.

1861 †WATSON, WILLIAM SPENCER, M.B., Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 7, Henrietta street, Cavendish square. C. 1883-4. *Trans.* 1.

1879 WATTEVILLE, ARMAND DE, M.A., M.D., B.Sc., Medical Electrician to St. Mary's Hospital; 30, Welbeck street, Cavendish square.

1854 WEBB, WILLIAM, M.D., Gilkin View House, Wirksworth, Derbyshire.

1840 WEBB, WILLIAM WOODHAM, M.D., 82, Avenue des Termes, Paris.

1842 †WEBER, FREDERIC, M.D., 44, Green street, Park lane. C. 1857. V.P. 1865.

1857 WEBER, HERMANN, M.D., Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. *Referee*, 1869-73, 1878-84. *Lib. Com.* 1864-73. *Trans.* 6.

1844 †WEGG, WILLIAM, M.D., 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873-80. *Lib. Com.* 1851-3.

1878 WEISS, HUBERT FOVEAUX, 11, Hanover square.

Elected.

1874 WELLS, HARRY, M.D., San Ysidro, Buenos Ayres, S. America.

1854 WELLS, SIR THOMAS SPENCER, Bart., Surgeon-in-Ordinary to H.M.'s Household; Consulting Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor street. C. 1870. V.P. 1881. *Trans.* 13. *Pro.* 1.

1842 †WEST, CHARLES, M.D., Corresponding Member of the Academy of Medicine of Paris; 29, Promenade des Anglais, Nice, Alpes Maritimes, France, and 2, Bolton row, Mayfair. C. 1855-6. V.P. 1863. P. 1877-8. *Referee*, 1848-54, 1857-62, 1864-76, 1880. *Sci. Com.* 1863. *Lib. Com.* 1844-7, 1851. *Trans.* 2.

1877 WEST, SAMUEL, M.D., Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; Physician to the Royal Free Hospital; Medical Registrar and Medical Tutor to St. Bartholomew's Hospital; 15, Wimpole street, Cavendish square. *Trans.* 2.

1882 WHARRY, CHARLES JOHN, M.D., Resident Superintendent, Government Civil Hospital, Hong Kong.

1881 WHARRY, ROBERT, M.D., 6, Gordon square.

1878 WHARTON, HENRY THORNTON, M.A., Surgeon to the Kilburn Dispensary; 39, St. George's road, Kilburn.

1828 WHATLEY, JOHN, M.D.

1875 WHIPHAM, THOMAS TILLYER, M.B., Physician to, and Lecturer on Clinical Medicine at, St. George's Hospital; 11, Grosvenor street, Grosvenor square.

1849 WHITE, JOHN.

1881 WHITE, WILLIAM HALE, M.D., Demonstrator of Anatomy at Guy's Hospital; 4, St. Thomas's street, Southwark.

1881 *WHITEHEAD, WALTER, F.R.S. Ed., Surgeon to the Manchester Royal Infirmary; Senior Surgeon to the Manchester and Salford Lock and Skin Hospital; 24, St. Ann's square, Manchester. *Trans.* 1.

1877 WHITMORE, WILLIAM TICKLE, 7, Arlington street, Piccadilly.

Elected

1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.

1870 *WILKIN, JOHN F., M.D., M.C., New Beckenham, Kent.

1883 *WILKINSON, THOMAS MARSHALL, Surgeon to the Lincoln County Hospital and to the Lincoln General Dispensary; 7, Lindum road, Lincoln.

1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.

1863 WILKS, SAMUEL, M.D., LL.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught; 72, Grosvenor street, Grosvenor square. *Referee*, 1872-81. *Sci. Com.* 1.

1883 *WILLANS, WILLIAM BLUNDELL, Great Hadham, Herts.

1865 †WILLETT, ALFRED, Surgeon to St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. C. 1880-81. *Referee*, 1882-4. *Trans.* 2.

1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.

1840 †WILLIAMS, CHARLES JAMES BLASIUS, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption, Brompton [47, Upper Brook street, Grosvenor square]; Villa de Rocher, Cannes. C. 1849-50. V.P. 1860-1. P. 1873-4. *Referee*, 1843-4. *Sci. Com.* 1862. *Trans.* 1.

1859 *WILLIAMS, CHARLES, Surgeon to the Norfolk and Norwich Hospital; 9, Prince of Wales road, Norwich.

1866 WILLIAMS, CHARLES THEODORE, M.A., M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 47, Upper Brook street, Grosvenor square. C. 1884. *Lib. Com.* 1880-3. *Trans.* 3.

1881 WILLIAMS, DAWSON, M.D., 4, Oxford and Cambridge Mansions, Marylebone road.

Elected

1872 WILLIAMS, JOHN, M.D., Assistant Obstetric Physician to University College Hospital; 11, Queen Anne street, Cavendish square. *Referee*, 1878-84. *Lib. Com.* 1876-82.

1868 WILLIAMS, WILLIAM RHYS, M.D., Commissioner in Lunacy; 19, Whitehall place.

1863 WILSON, ROBERT JAMES, 7, Warrior square, St. Leonard's-on-Sea, Sussex.

1850 *WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Beech Lawn, Banbury.

1825 WISE, THOMAS ALEXANDER, M.D., Inchrye Abbey, New borough, Fife, N.B.

1879 WOAKES, EDWARD, M.D., Senior Aural Surgeon to the London Hospital; 78, Harley street, Cavendish square.

1851 †WOOD, JOHN, F.R.S., Professor of Clinical Surgery in King's College, London, and Senior Surgeon to King's College Hospital; Examiner in Surgery in the University of London; 61, Wimpole street, Cavendish square. C. 1867-8. V.P. 1877-8. *Referee*, 1871-6, 1880-84. *Lib. Com.* 1866. *Trans.* 3.

1848 †WOOD, WILLIAM, M.D., Physician to St. Luke's Hospital for Lunatics; 99, Harley street, Cavendish square. C. 1867-8. V.P. 1877-8.

1883 WOOD, WILLIAM EDWARD RAMSDEN, M.A., M.D., Assistant Medical Officer, Bethlem Royal Hospital, St. George's road, Southwark.

1881 WOODMAN, SAMUEL, Consulting Surgeon to the Ramsgate and St. Lawrence Royal Dispensary; 5, Prospect terrace, Ramsgate.

1879 WOODWARD, G. P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.

1842 WORTHINGTON, WILLIAM COLLINS, London road, Lowestoft, Suffolk. *Trans.* 3.

Elected

1865 WOTTON, HENRY, M.D., 15, Notting Hill terrace, Kensington.

1878 YEO, GERALD F., M.D., M.C., Professor of Physiology in King's College, London; Examiner in Physiology, University of London; King's College, Strand.

[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1883 CARPENTER, WILLIAM BENJAMIN, C.B., M.D., LL.D., F.R.S., Corresponding Member of the Institute; 56, Regent's park road.

1847 CHADWICK, EDWIN, C.B., Corresponding Member of the Academy of Moral and Political Sciences of the Institute of France; Park Cottage, East Sheen.

1883 FRANKLAND, EDWARD, M.D., D.C.L., Ph.D., F.R.S., Professor of Chemistry in the Royal School of Mines; Corresponding Member of the French Institute; Royal College of Chemistry, South Kensington Museum, and the Yews, Reigate Hill, Reigate.

1868 HOOKER, SIR JOSEPH DALTON, C.B., M.D., K.C.S.I., D.C.L., LL.D., F.R.S., Member of the Senate of the University of London, Director of the Royal Botanic Gardens, Kew; Corresponding Member of the Academy of Sciences of the Institute of France; Royal Gardens, Kew.

1868 HUXLEY, THOMAS HENRY, LL.D., F.R.S., Professor of Natural History in the Royal School of Mines; Secretary to the Royal Society; Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, Dresden, &c.; 4, Marlborough place, St. John's wood.

1878 LUBBOCK, SIR JOHN, Bart., M.P., D.C.L., LL.D., F.R.S., High Elms, Bromley, Kent.

Elected

1847 OWEN, SIR RICHARD, K.C.B., D.C.L., LL.D., F.R.S., late Superintendent of the Natural History Departments in the British Museum; Foreign Associate of the Academy of Sciences of the Institute of France; Sheen Lodge, Mortlake.

1883 PARKER, WILLIAM KITCHEN, F.R.S., Hunterian Professor of Comparative Anatomy in the Royal College of Surgeons; 36, Claverton street, Pimlico.

1873 STOKES, GEORGE GABRIEL, M.A., D.C.L., LL.D., Lucasian Professor of Mathematics in the University of Cambridge; Secretary to the Royal Society, &c.; Lensfield Cottage, Cambridge.

1868 TYNDALL, JOHN, D.C.L., LL.D., F.R.S., Professor of Natural Philosophy in the Royal Institution; Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, Geneva, &c.; Royal Institution, Albemarle street, Piccadilly.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1878 BACCELLI, GUIDO, M.D., Professor of Medicine at Rome.

1883 BIGELOW, HENRY J., M.D., Professor of Surgery at Harvard University, and Surgeon to the Massachusetts General Hospital.

1876 BILLROTH, THEODOR, M.D., Professor of Surgery in the University of Vienna; Vienna.

1883 CHARCOT, J. M., M.D., Physician to the Hôpital de la Salpêtrière, and Professor at the Faculty of Medicine of Paris; Member of the Academy of Medicine; Quai Malaquais 17, Paris.

1864 DONDERS, FRANZ CORNELIUS, M.D., LL.D., Professor of Physiology and Ophthalmology at the University of Utrecht.

1883 DUBOIS REYMOND, EMIL, M.D., Professor in Berlin; N. W. Neue Wilhelmstrasse 15, Berlin.

1876 EDWARDS, H. MILNE, M.D., Member of the Institute of France, and of the Academy of Medicine; Dean of the Faculty of Sciences and Professor at the Museum of Natural History of Paris; 57, Rue Cuvier, Paris.

1878 GUENEAU DE MUSSY, NOEL, M.D., Member of the Academy of Medicine; Physician to the Hôtel Dieu; 4, Rue St. Arnaud, Paris.

1866 HANNOVER, ADOLPH, M.D., Professor at Copenhagen.

Elected

1873 HELMHOLTZ, HERMANN LUDWIG FERDINAND, Professor of Physics and Physiological Optics; Berlin.

1859 HENLE, J., M.D., Professor of Anatomy at Göttingen.

1873 HOFMANN, A. W., LL.D., Ph.D., Professor of Chemistry, Berlin.

1868 KÖLLIKER, ALBERT, Professor of Anatomy in the University of Würzburg.

1856 LANGENBECK, BERNHARD, M.D., late Professor of Surgery in the University of Berlin.

1868 LARREY, HIPPOLYTE BARON, Member of the Institute of France; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées;" Commander of the Legion of Honour, &c.; Rue de Lille, 91, Paris.

1883 PASTEUR, LOUIS, LL.D., Member of the Institute of France (Academy of Sciences).

1878 SCANZONI, FRIEDREICH WILHELM VON, Royal Bavarian Privy Councillor, and Professor of Medicine in the University of Würzburg.

1856 VIRCHOW, RUDOLPH, M.D., LL.D., Professor of Pathological Anatomy in the University of Berlin; Corresponding Member of the Academy of Sciences of the Institute of France; Berlin.

LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

DATE OF ELECTION.

1819 Jas. M. Arnott, F.R.S.
1833 Sir George Burrows, Bt., M.D., F.R.S.
Thomas A. Barker, M.D.
1835 Richard Quain, F.R.S.
Thomas A. Nelson, M.D.
1836 Alexander Shaw.
J. George French.
1837 Thomas Blizard Curling, F.R.S.
George Busk, F.R.S.
1838 Charles Hawkins.
Henry Spencer Smith.
1839 T. Graham Balfour, M.D., F.R.S.
Fred. Le Gros Clark, F.R.S.
James Dixon.
1840 Chas. J. B. Williams, M.D., F.R.S.
Charles Hutton, M.D.
Samuel A. Lane.
Sir James Paget, Bt., F.R.S.
1841 Sir Henry A. Pitman, M.D.
William Bowman, F.R.S.
John Parkin, M.D.
Paul Jackson.
1842 Charles West, M.D.
Frederic Weber, M.D.
John Simon, C.B., F.R.S.
John Erichsen, F.R.S.
Sir Oscar M. P. Clayton.
1843 Robert Greenhalgh, M.D.
Sir Prescott G. Hewett, Bt., F.R.S.
Henry Lee.
Wm. White Cooper.
Luther Holden.
Edward Newton.
1844 Arthur Farre, M.D., F.R.S.
1844 William Wegg, M.D.
Thomas King Chambers, M.D.
Edwin Humby.
1845 Samuel Cartwright.
George D. Pollock.
Thomas Taylor.
Sir Edwin Saunders.
William Oliver Chalk.
Edward U. Berry.
Benjamin Ridge, M.D.
1846 John A. Bostock.
Barnard Wight Holt.
Carsten Holthouse.
1847 W. H. O. Sankey, M.D.
George Johnson, M.D., F.R.S.
1848 Edward H. Sieveking, M.D.
Edward Ballard, M.D.
William Wood, M.D.
Thomas Hawksley, M.D.
Edward John Tilt, M.D.
John Clarke, M.D.
John Gay.
John Gregory Forbes.
1849 Hugh J. Sanderson, M.D.
C. H. F. Routh, M.D.
Edmund L. Birkett, M.D.
George T. Fincham, M.D.
Sir William W. Gull, Bt., M.D., F.R.S.
1850 Richard Quain, M.D., F.R.S
George Roper, M.D.
1851 Sir Wm Jenner, Bt., M.D., F.R.S.
H. Haynes Walton.
John Birkett.
John A. Kingdon.

1851 Peter Y. Gowland.
John Marshall, F.R.S.
John Wood, F.R.S.
Bernard E. Brodhurst.
Robert J. Spitta, M.D.
George Gaskoin.
1852 C. Bland Radcliffe, M.D.
Walter H. Walshe, M.D.
William Adams.
John Cooper Forster.
Sir Henry Thompson.
1853 Robert Brudenell Carter.
1854 Alfred Baring Garrod, M.D., F.R.S.
Samuel O. Habershon, M.D.
Sir Thomas Spencer Wells, Bt.
1855 W. M. Graily Hewitt, M.D.
J. Burdon Sanderson, M.D., F.R.S.
J. Russell Reynolds, M.D., F.R.S.
Walter John Bryant, M.D.
1856 Charles J. Hare, M.D.
William Bird.
Jonathan Hutchinson, F.R.S.
Timothy Holmes.
Alonzo H. Stocker, M.D.
1857 William Overend Priestley, M.D.
George Harley, M.D., F.R.S.
Henry Thompson, M.D.
Hermann Weber, M.D.
George Owen Rees, M.D., F.R.S.
John Whitaker Hulke, F.R.S.
John Morgan.
Henry Cooper Rose, M.D.
Henry Walter Kiallmark.
1858 Fred. George Reed, M.D.
William Chapman Begley, M.D.
John William Ogle, M.D.
Wilson Fox, M.D., F.R.S.
John Fremlyn Streatfeild.
Francis Harris, M.D.
1859 Wm. Howship Dickinson, M.D.
William Scovell Savory, F.R.S.
Edwin Thomas Truman.
Francis Hird.
Richard Barwell.
Edward Tegart.
Septimus William Sibley.
William E. Stewart.
1860 Sir Andrew Clark, Bt., M.D.
John Maclean, M.D.
Sigismund Sutro, M.D.
William Ogle, M.D.
Thomas Bryant.
John Couper.
Henry Howard Hayward.
1861 Robert Barnes, M.D.
William Spencer Watson.
William Henry Holman.
1862 James Andrew, M.D.
Lionel Smith Beale, M.B., F.R.S.
Thomas H. Tuke, M.D.
Edmund Symes Thompson, M.D.
Reginald Edward Thompson, M.D.
William Henry Brace, M.D.
George Cowell.
M. Berkeley Hill.
1863 Octavius Sturges, M.D.
John Langdon H. Down, M.D.
Samuel Wilks, M.D., F.R.S.
Samuel Fenwick, M.D.
Julius Althaus, M.D.
Sydney Ringer, M.D.
Thomas Smith.
Arthur B. R. Myers.
Arthur E. Durham.
William Sedgwick.
1864 George Buchanan, M.D., F.R.S.
Charles Derby Waite, M.B.
John Harley, M.D.
Walter John Coulson.
Thomas William Nunn.
Francis Mason.
Jos. Gillman Barratt, M.D.
1865 Charles Robert Drysdale, M.D.
James Edward Pollock, M.D.
William Cholmeley, M.D.
Reginald Southey, M.D.
George Fielding Blandford, M.D.
Dyce Duckworth, M.D.
Frederick W. Pavy, M.D., F.R.S.
William Morrant Baker.
John Langton.
Frederick James Gant.
Alfred Willett.
Bowater John Vernon.
Alfred Cooper.
Christopher Heath.
Henry Wotton.
1866 Thomas Fitzpatrick, M.D.
Samuel Jones Gee, M.D.
Charles Theodore Williams, M.D.
Heywood Smith, M.D.
John Crockett Fish, M.D.
William Selby Church, M.D.
Edward John Waring, M.D.
1867 William Henry Day, M.D.
Achille Vintras, M.D.
Richard Douglas Powell, M.D.
F. Howard Marsh.

1867 Henry Power.
Sir William MacCormac.
Thomas Pickering Pick.
John Astley Bloxam.
Charles Arthur Aikin.
Samuel Hill, M.D.

1868 H. Charlton Bastian, M.D., F.R.S.
William Henry Broadbent, M.D.
Thomas Buzzard, M.D.
John Cavafy, M.D.
Walter Butler Cheadle, M.D.
John Cockle, M.D.
Thomas Crawford, M.D.
T. Henry Green, M.D.
William Rhys Williams, M.D.
Walter Moxon, M.D.
William Chapman Grigg, M.D.
John Croft.
George Eastes.
William Henry Freeman.

1869 Joseph Frank Payne, M.D.
Arthur E. Sansom, M.D.
John Wickham Legg, M.D.
Charles Elam, M.D.
Thomas Laurence Read.

1870 Alfred Meadows, M.D.
William Wadham, M.D.
J. Warrington Haward.
Edgcombe Venning.
Clement Godson, M.D.

1871 William Cayley, M.D.
Charles Henry Ralfe, M.D.
Arthur Julius Pollock, M.D.
Thomas L. Brunton, M.D., F.R.S.
Henry Gawen Sutton, M.D.
J. Hughlings Jackson, M.D., F.R.S.
Henry Sutherland, M.D.
George Vivian Poore, M.D.
Walter Rivington.
Marcus Beck.
Edward Bellamy.
William F. Butt.
Benjamin Duke.

1872 Gilbart Smith, M.D.
Thomas B. Christie, M.D.
George B. Brodie, M.D.
John Williams, M.D.
Sir J. Fayrer, M.D., F.R.S.
Charles S. Tomes, B.A., F.R.S.
William Bartlett Dalby.

1873 William Miller Ord, M.D.
Frederick Taylor, M.D.
Norman Moore, M.D.
John Curnow, M.D.

1873 William R. Gowers, M.D.
William Guyer Hunter, M.D
Charles Creighton, M.D.
Jeremiah McCarthy.
Wm. Johnson Smith.
Robert William Parker.
Alex. O. McKellar.
Henry T. Butlin.
Charles Higgens.
William J. Walsham.
Edward Milner.

1874 Alfred Lewis Galabin, M.D.
George Thin, M.D.
Alfred B. Duffin, M.D.
James H. Aveling, M.D.
Evan B. Baxter, M.D.
John M. Bruce, M.D.
Henry Morris.
William Laidlaw Purves.
William Harrison Cripps.
Henry G. Howse.
Herbert William Page.
Frederic Durham.
John J. Merriman.

1875 Thomas T. Whipham, M.B.
Francis Charlewood Turner, M.D
Herbert Tibbits, M.D.
Robert Hunter Semple, M.D.
Thomas Crawford Hayes, M.D.
Charles Henry Carter, M.D
Fletcher Beach, M.B
Samuel Osborn.
Waren Tay.
Edmund J. Spitta.

1876 Thomas Barlow, M.D.
John C. Bucknill, M.D., F.R.S.
Wm. Lewis Dudley, M.D.
Albert J. Venn, M.D.
John Knowsley Thornton.
Charles Macnamara.
John N. C. Davies-Colley.

1877 Felix Semon, M.D.
Sidney Coupland, M.D.
Francis Warner, M.D.
William Ewart, M.D.
Alfred Pearce Gould.
J. Rickman Godlee.
Alban H. G. Doran.
George Ernest Herman, M.B.
Samuel West, M.D.
John Abercrombie, M.B.
J. Matthews Duncan, M.D., F.R.S.
Henry de Fonmartin, M.D.

1877 George Allan Heron, M.D.
Joseph A. Ormerod, M.D.
P. Henry Pye-Smith, M.D.
Edward Nettleship.
William Henry Bennett.
Benj. T. Lowne.
William T. Whitmore.
1878 Jas. Crichton Browne, M.D.
Fred. T. Roberts, M.D.
Joseph Lister, F.R.S.
Clinton T. Dent.
John H. Morgan.
Walter Pye.
Gerald F. Yeo, M.D.
Donald W. Charles Hood, M.B.
Henry Gervis, M.D.
Herbert Watney, M.D.
Richard Davy.
Hubert Foveaux Weiss.
Henry Thornton Wharton.
1879 Alfred Sangster, M.B.
Edward Woakes, M.D.
Armand de Watteville, M.D.
Malcolm A. Morris.
A. E. Cumberbatch.
Edmund Owen.
Arthur E. J. Barker.
Frederick Treves.
Horatio Donkin, M.B.
Thomas John Maclagan, M.D.
David White Finlay, M.D.
Andrew Clark.
S. Hamilton Cartwright.
John H. Waters, M.D.
Francis Henry Champneys, M.B.
William Watson Cheyne.
William Munk, M.D.
George Henry Savage, M.D.
H. H. Clutton, M.A.
Frederic S. Eve.
E. Noble Smith.
William Henry Allchin, M.B
F. G. Dawtrey Drewitt, M.D.
1880 Robert Alex. Gibbons, M.D.
David Ferrier, M.D., F.R.S.
Vincent Dormer Harris, M.D.
Edmund D. Maddick.
Jas. John MacWhirter Dunbar, M.B.
James William Browne, M.B.
William Appleton Meredith, M.B.
Alexander Hughes Bennett, M.D.
Malcolm Macdonald McHardy.
A. Boyce Barrow.
William Murrell, M.D.
1880 Bernard O'Connor, A.B., M.D.
Leslie Ogilvie, M.B.
George Lockwood Laycock, M.B.
George Ogilvie, M.B.
Charles Edward Beevor, M.D.
Thomas Colcott Fox, M.B.
George Henry Makins.
1881 Francis de Havilland Hall, M.D.
Robert Wharry, M.D.
Cecil Yates Biss, M.D.
Richard Clement Lucas.
Stephen Mackenzie, M D.
James Anderson, M.D..
William Hale White, M.D.
Eustace Smith, M.D.
William Sinclair Thomson, M.D.
Percy Kidd, M.D.
Oswald A. Browne, M.A.
Audley Cecil Buller.
W. Bruce Clarke, M.B.
Dawson Williams, M.D.
George Lindsay Johnson, M.A., M.D.
Henry Edward Juler.
Jonathan F. C. H. Macready.
Henry Howard, M.B.
Samuel Nall, M.B.
C. B. Lockwood.
1882 Philip J. Hensley, M.D.
Isidore I. Lyons.
John Barclay Scriven.
Yelverton Dawson, M.D.
Charles S. Roy, M.D.
George Robertson Turner.
Howard Henry Tooth, M.B.
Herbert Isambard Owen, M.D.
Charles R. B. Keetley.
Joseph Mills.
A. T. Myers, M.D.
Anthony A. Bowlby.
Amand J. McC. Routh, M.D.
Seymour J. Sharkey, M.B.
William Lang.
Henry Radcliffe Crocker, M.D.
William Edward Steavenson, M.B.
D. Astley Gresswell, M.B.
1883 Edwin Clifford Beale, M.A., M.B.
James Kingston Fowler, M.D.
James Frederic Goodhart, M.D.
John Charles Galton, M.A.
Walter Hamilton Acland Jacobson.
Edward Joshua Edwardes, M.D.
Walter H. H. Jessop, M.B.
Walter Edmunds, M.D.

1883 William E. Ramsden Wood, M.D.
Victor A. Horsley.
Dudley Wilmot Buxton, M.D.
Charles Douglas F. Phillips, M.D.
Hutchinson Royes Bell.
Angel Money, M.D.
John James Pringle, M.B.
Henry Roxburgh Fuller, M.B.
Wilmot Parker Herringham, M.B.
John Bland Sutton.
James Dixon Bradshaw, M.B.
Robert Marcus Gunn, M.B.
Storer Bennett.
Henry Maudsley, M.B.
William Pasteur, M.B.
William Rose, M.B.
Edward Albert Schäfer, F.R.S.

1884 Augustus Waller, M.D.
Stanley Boyd, M.B.
Charles Stonham.
William Arbuthnot Lane, M.S.
George Newton Pitt, M.D.
Frederic Bowreman Jessett.
George Lawson.
Arthur Marmaduke Sheild, M.B.
Wayland Charles Chaffey, M.B.
Dennis Dellaway.
Heneage Gibbes, M.D.
Sidney Harris Cox Martin, M.B.
Thomas Whitehead Reid.
William P. Huggard, M.D.
Lionel Druitt, M.D.
Robert James Lee, M.D
Thomas Wakley, Jun.

CONTENTS.

	PAGE
List of Officers and Council	v
List of Presidents of the Society	vi
Referees of Papers	vii
Trustees of the Society	viii
Trustees of the Marshall Hall Memorial Fund	viii
Library Committee	viii
List of Fellows	ix
List of Honorary Fellows	lxvii
List of Foreign Honorary Fellows	lxix
List of Resident Fellows, arranged according to Date of Election	lxxi
List of Plates	lxxxi
Woodcuts	lxxxii
Advertisement	lxxxiii
Regulations relative to Proceedings	lxxxiv

I. Address of JOHN MARSHALL, F.R.S., President, at the Annual Meeting, March 1st, 1884 . . 1

II. Cerebral Localisation. A Review and Forecast. Being the Marshall Hall Prize Oration. By DAVID FERRIER, M.D., LL.D., F.R.S. . . . 33

III. Case of Spontaneous Inguinal Aneurism in a boy aged twelve years; for which the external iliac artery was tied. With a table of all the other recorded cases of external aneurism in persons under twenty years of age. By ROBERT WILLIAM PARKER, M.R.C.S. Eng., Surgeon to the East London Hospital for Children . . . 51

IV. Investigations into the action of the Digitalis Group. By SYDNEY RINGER, M.D., F.R.C.P., Professor of Medicine at University College, London, and HARRINGTON SAINSBURY, M.D., M.R.C.P., Physician to the North-West London Hospital . . 67

PAGE

V. On High Amputation for Senile Gangrene. By JONATHAN HUTCHINSON, F.R.S., Emeritus Professor of Surgery to the London Hospital, and President of the Ophthalmological Society . 91

VI. Addendum to third communication on Artificial Respiration in Still-born Children. (Mediastinal Emphysema and Pneumothorax in connection with Tracheotomy. An experimental inquiry.) By FRANCIS HENRY CHAMPNEYS, M.A., M.B. Oxon., F.R.C.P., Assistant Lecturer on Midwifery, and Assistant Obstetric Physician to St. George's Hospital 101

VII. Fourth communication on Artificial Respiration in Still-born Children. Certain minor points. An experimental inquiry. By FRANCIS HENRY CHAMPNEYS, M.A., M.B. Oxon., F.R.C.P., Assistant Lecturer on Midwifery, and Assistant Obstetric Physician to St. George's Hospital . . . 105

VIII. The direct treatment of Psoas Abscess with Caries of the Spine. By FREDERICK TREVES, F.R.C.S., Assistant Surgeon to, and Lecturer on Anatomy at, the London Hospital 113

IX. Radical cure of a large Spina Bifida in an Adult. By WALTER WHITEHEAD, F.R.C.S.E., F.R.S. Edin., Surgeon, Manchester Royal Infirmary; Senior Surgeon, Manchester and Salford Lock and Skin Hospital 127

X. On a form of Alcoholic Spinal Paralysis. By W. H. BROADBENT, M.D., F.R.C.P., Lecturer on Medicine at, and Physician to, St. Mary's Hospital . . 133

XI. Neurectomy of the Second Division of the Fifth Nerve. By THOMAS F. CHAVASSE, M.D., C.M. (Edin.), Surgeon to the Birmingham General Hospital 145

XII. On the value of Systematic Examination of Stillborn Children. By JOHN BLAND SUTTON, Lecturer on Comparative Anatomy and Senior Demonstrator of Anatomy, Middlesex Hospital Medical College . 157

PAGE

XIII. Three cases of Xeroderma Pigmentosum (Kaposi) or Atrophoderma Pigmentosum. By H. RADCLIFFE CROCKER, M.D., M.R.C.P., Physician to the Skin Department of University College Hospital; Physician to the East London Hospital for Children . 169

XIV. The Pathology of Myxœdema as illustrated in a typical case. By JOHN HARLEY, M.D. Lond., F.R.C.P., Lecturer on General Anatomy and Physiology and Physician to St. Thomas's Hospital . 189

XV. An average or "Composite" Temperature Chart of Smallpox. By G. ERNEST HERMAN, M.B. Lond., M.R.C.P. Lond., F.R.C.S. Eng., Obstetric Physician to, and Lecturer on Midwifery at, the London Hospital, &c. 205

XVI. A case of Gangrene of the Lung following Necrosis of the Temporal Bone, the result of Scarlet Fever. Treatment by Drainage. Recovery. By W. CAYLEY, M.D., Physician to, and Lecturer on Medicine at, the Middlesex Hospital; Physician to the London Fever Hospital, &c.; and A. Pearce Gould, M.S., Assistant Surgeon to the Middlesex Hospital, &c. . 209

XVII. On the treatment of Pus-secreting Basic Cavities of the Lung by the method of Paracentesis and Free Drainage. By CECIL Y. BISS, M.A., M.D. (Cantab.), M.R.C.P., Assistant Physician to the Hospital for Consumption, Brompton, and the Middlesex Hospital 217

XVIII. Three forms of Spinal Deformity. By W. ARBUTHNOT LANE, M.S., F.R.C.S., Demonstrator of Anatomy to Guy's Hospital, and Assistant Surgeon to the Hospital for Sick Children, Great Ormond Street 233

XIX. On a particular form of Amnesia. Loss of nouns. By W. H. BROADBENT, M.D., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the London Fever Hospital . . 249

XX. Embolism of the Right Middle Cerebral Artery, producing Left Hemiplegia and Hemianæsthesia: absorption of a large portion of the Right Hemisphere: death seven years later. By SEYMOUR J. SHARKEY, M.B., Assistant Physician to St. Thomas's Hospital 265

PAGE

XXI. On Albuminuria with Parametritis. By J. Matthews Duncan, M.D., F.R.S., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Bartholomew's Hospital . . 273

XXII. On the Weight of the Thyroid Body in persons dying from various causes. By Stephen Mackenzie, M.D., F.R.C.P., Physician to, and Lecturer on Medicine at, the London Hospital . . . 277

Index 281

LIST OF PLATES

PAGE

I. On the Value of Systematic Examination of Stillborn Children. (J. B. SUTTON.) Fig. 1. Pulmonary orifice of the heart of a fœtus. Fig. 2. Left ventricle of the same heart. Fig. 3. Posterior view of the skull of a human fœtus at the end of the fourth month. Fig. 4. Ditto at the end of the third month. Fig. 5. External configuration of the fœtus represented on Plate II . 168

II. Ditto. Fig. 1. Lateral curvature of the spine in a human fœtus. Fig. 2. The skeleton of the dwarfed limb from the same case 168

III. Three Cases of Xeroderma Pigmentosum (Kaposi) or Atrophoderma Pigmentosum. (H. RADCLIFFE CROCKER, M.D.) Elizabeth Beddoes . . 188

IV. Ditto. Benjamin Beddoes 188

V. Ditto. Figs. 1—3. Microscopical drawings of tumours . 188

VI. An Average or "Composite" Temperature Chart of Smallpox. (G. ERNEST HERMAN, M.B.) . . 208

VII. A Case of Gangrene of the Lung, treated by Drainage. (W. CAYLEY and A. PEARCE GOULD.) Temperature chart 216

VIII. Embolism of Right Middle Cerebral Artery. (S. J. SHARKEY, M.B.) Fig. 1. View of the vertex of the brain. Fig. 2. View of the base of the brain . 271

IX. Ditto. Fig. 1. Lateral view of left hemisphere. Fig. 2. Lateral view of right hemisphere . . . 272

Woodcuts.

PAGE

1 and 2. Spontaneous Inguinal Aneurism in a Boy. (R. W. Parker.) Sphygmographic tracings . . 53

3. Ditto. (*a*) Seat of ligature; (*b*) aneurysmal sac; (*c*) anterior crural nerve; (*d*) external incision, through which the artery was approached . . . 66

4 and 5. Radical Cure of a Large Spina Bifida in an Adult. (W. Whitehead.) Before and after operation . 129

6. On the Value of Systematic Examination of Stillborn Children. (J. B. Sutton.) Fig. 1. Complete stricture of the duodenum. Fig. 2. The *cul-de-sac* inverted, showing a band of muscular fibre 166

7. On the Treatment of Pus-secreting Basic Cavities of the Lung by Paracentesis and Free Drainage. (C. Y. Biss.) Chest diagram 220

8 and 9. Ditto ditto 223

10, 11, 12, 13. Three forms of Spinal Deformity. (W. Arbuthnot Lane) 238, 243, 245

ADVERTISEMENT.

THE Council of the Royal Medical and Chirurgical Society deems it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, or opinions set forth in the various papers which, on grounds of general merit, are thought worthy of being published in its 'Transactions.'

Regulations relative to the publication of the 'Proceedings of the Society.'

That, as a general rule, the 'Proceedings' will be issued every two months, subject to variations dependent on the extent of matter to be printed.

That a Copy of the 'Proceedings' will be sent, postage free, to every Fellow of the Society resident in the United Kingdom.

That the 'Proceedings of the Society' may be obtained by non-members at the Society's House, 53, Berners Street, on pre-payment of an annual subscription of five shillings, which may be transmitted either by post-office order or in postage-stamps; —this will include the expense of conveyance by post to any part of the United Kingdom; to other places they will be sent, carriage free, through a bookseller, or by post, the receiver paying the foreign charges.

That a notice of every paper will appear in the 'Proceedings.' Authors will be at liberty, on sending their communications, to intimate to the Secretary whether they wish them to appear in the 'Proceedings' *only*, or in the 'Proceedings' and 'Transactions;' and in all cases they will be expected to furnish an Abstract of the communication.

The Abstracts of the papers read will be furnished to the Journals as heretofore.

ADDRESS

OF

JOHN MARSHALL, F.R.S.

PRESIDENT,

AT THE

ANNUAL MEETING, MARCH 1ST, 1884.

GENTLEMEN,—FELLOWS OF THE ROYAL MEDICAL AND CHIRURGICAL SOCIETY,—Referring you to the Annual Report of the Council for information concerning the general and financial condition of the Society, I once more, and now for the last time, address myself to the unwelcome duty of alluding to the gaps which have been made by death in the ranks of the Society, during the official year which has just closed in upon us.

Amongst our *non-resident* Fellows, seven have died, viz.: Drs. Hudson, Evans, Iles, Marion Sims, Macnaught, Gully, and Harding. Eight *resident* Fellows have disappeared from our list, viz.: Drs. Meredyth, Hilton Fagge, Robert Druitt, Patrick Stewart, and Jones; and Messrs. Harper, Phillips, and Montefiore. We have lost but one *Honorary* Fellow, Dr. Wm. Farr.

In speaking of the professional careers of these recently deceased non-resident and resident colleagues, I will follow, as on the like occasion last year, an order corresponding with the length of time during which they have

been numbered amongst us; and I, again, acknowledge my great indebtedness to various periodicals for the facts which I am able to set forth in the following brief narratives.

Robert Samuel Hudson, the first of the *non-resident* Fellows to be mentioned, practised at Redruth in Cornwall. He had been a Fellow for only six years. He was medically educated, partly in Belfast and partly in Dublin. He became a Licentiate of the Royal College of Surgeons of Ireland, and a Doctor of Medicine of the Queen's University in 1868. In 1880, he was made a Fellow of the Irish College, by examination. His activity as a member of our profession was shown by his occasional contributions to the Medical journals, by his having been chosen President of the South Western Branch of the British Medical Association, and by his having established a local branch of the St. John's Ambulance Society. He also assisted in instituting Popular Science Lectures in Redruth. His death took place last autumn in Ireland, at the comparatively early age of forty years.

George Henry Evans, M.A. and M.D. of Cambridge (1871), entered our profession late in life. He was a Fellow of this Society for nine years. He had been trained partly at Addenbroke's, but chiefly and lastly at St. Thomas's Hospital. His thesis on "Paracentesis thoracis," full of elaborate statistics, is published in the 'Reports' of that Institution (V. ii, New Series). Having become an M.R.C.P. in 1871, Dr. Evans was appointed Assistant Physician to Middlesex Hospital (1874). He had special charge of the Skin Department for some time, and a Paper by him, on "A case of an eruption supposed to be the 'acne cachecticorum' of Hebra," was published in our 'Proceedings' (V. viii). He was also Lecturer on Public Health. A man of high culture and varied attainments, an able Physician, and a genial colleague, he was compelled to retire as far

back as 1878. He died of general paralysis last September, at the age of forty-eight years.

Francis Henry Wilson Iles, of Watford, Herts, died in that town, also in September, at the age of forty-nine years, from the effects of blood-poisoning, contracted about a month previously during the performance of the operation of tracheotomy. He had belonged to our Society for seventeen years. A native of Lincolnshire, he studied at St. George's Hospital and Medical School, and also in Dublin and Paris. In 1855, having taken the Licentiateship of the Society of Apothecaries and the Membership of the Royal College of Surgeons, he was appointed Resident Medical Officer to the Western General Dispensary, to which Institution he acted as Surgeon during the cholera epidemic of that period. In 1859, he became a partner in the practice of Dr. Pidcock, at Watford. In 1862, he acquired the degree of M.D. at Aberdeen. For more than twenty years, he was a successful and much esteemed practitioner and an active townsman, supporting in a spirited manner all kinds of local improvements. He was Surgeon to the West Hertford Infirmary, and Honorary Assistant Surgeon to the 2nd Hertfordshire Volunteers.

James Marion Sims, the well-known American Gynæcologist, was a non-resident Fellow of this Society for eighteen years. He was born in 1813, in South Carolina, and died in New York, on the 18th November last year, in the seventy-first year of his age.

Having graduated at the South Carolina College, he proceeded to study medicine at first in Charleston, but subsequently at the Jefferson Medical College in Philadelphia, from which institution he proceeded M.D. in 1835. In the following year, at the age of twenty-two, he commenced practice in Montgomery, Alabama, where he speedily acquired a considerable reputation as a general Surgeon; but he quickly availed himself of certain special conditions met with in that locality. The very frequent occurrence of vesico-vaginal fistula in the young and pro-

bably imperfectly assisted negresses on the neighbouring plantations, turned his attention to that injury, and induced him to make persevering attempts to cure it by operation. After having repeatedly failed, as he concluded, through the use of silk sutures, he tried silver wire, and then met with great success. By further improvements in many details,—as in the form of the speculum employed, and in the modes of paring the edges of the wound, of introducing and securing the silver wires, of supporting the sides of the wound, and of ensuring the constant evacuation of the urinary bladder,—he at last obtained the most brilliant results. Other Surgeons had, no doubt, preceded him in the performance of this operation, and some of his coadjutors, more especially Dr. Bozeman, shared the merit of introducing certain important improvements in it; but from his great ingenuity, his unremitting perseverance, and his manual dexterity, his personal achievements were pre-eminent, and his name has been and will continue to be especially remembered in connection with this great triumph of the Surgical art.

Having moved, in 1853, from Montgomery to New York, Dr. Sims practised for about a dozen years in the last-named city, during which period, however, he visited Europe; and he so frequently expounded and demonstrated his mode of operating, at different hospitals in Dublin, London, Brussels, and Paris, and was so frequently consulted by private patients, that he became recognised as almost a European practitioner.

It was not alone by this one operation that Dr. Sims gained his widely-spread reputation, or conferred the greatest benefit on womankind. It was through his instrumentality that the New York State Women's Hospital was founded. He was its first Physician, and he contributed largely to the acknowledged efficiency of that important Institution, which has, indeed, formed a centre from which so much and such beneficial scientific and practical knowledge has been diffused through the whole of the United States. From his labours in this field, Dr.

Sims has been credited with having led the way to the establishment of a special department of practice, viz. Gynæcology, as distinguished from Obstetrics proper, or pure Midwifery.

During and after the great civil strife in America, Dr. Sims' Southern feelings may have been somewhat tried. Some tension also occurred in his relations with his hospital colleagues. No doubt, temptations likewise arose to extend his fame and his practice in Europe. He left New York for London in 1865, and remained amongst us until 1868, occasionally visiting the Continent. During the rest of his active life, he may be said to have migrated between New York, London, Brussels, Paris, Nice, and Rome. It is a singular episode in his career that in 1870, at the age of fifty-seven, he left Paris as Chief Surgeon to the Anglo-American Ambulance Corps, which did such good service at Sedan, in helping to heal some of the physical injuries inflicted in the Franco-German war. I am here reminded that others of our Fellows—I will venture to particularise Sir Wm. MacCormac and Mr. Marcus Beck—also lent their Surgical aid in that great struggle.

It was in 1881, during one of his temporary halts in London, that Dr. Sims had a very dangerous attack of pneumonia, through which he was diligently and safely guided by another of our Fellows, Dr. William Ord. So recently as last August, he was again in London, in seemingly good health. In November, he was about to leave New York to pass the winter in Italy, but was induced to remain a week or so longer than he intended, in order to undertake an operation. Having complained one evening of pain in the region of the heart, he died from an attack of angina in the course of the ensuing night.

It is worthy of note that Dr. Sims never held any post as a Lecturer or Professor, and certainly his pen was not so prolific as might have been expected. In early life, he published a paper on "Trismus Nascentium;" after that, a defence of the "Silver Wire Sutures;" then essays

on "Ovariotomy," on "Intra-Uterine Fibrous Tumours," and on the "Use of the Microscope in the Study of Uterine Diseases;" comparatively recently, an account of "A Case of Cholecystotomy" (an operation which he himself was amongst the first to perform); and lastly, a contribution on the "Treatment of Gunshot Wounds of the Abdomen." His most original work, entitled 'Clinical Notes on Uterine Surgery,' requires special remark. It abounds in valuable matter and suggestions; but the proposal which it contains, to treat sterility in women by artificial impregnation, gave a shock to social, legal, and medical susceptibilities, as involving questions of social propriety, of possible fraud, and of professional self-respect. His long-established general and special reputation survived this doubtful recommendation.

Dr. Sims was honoured by decorations from many European Governments; he received titles from numerous American and European Scientific and Medical societies; and in 1876, he was elected President of the American Medical Association. His personal appearance and genial manners impressed most people favorably towards him. He was of a warm temper, but not of an irreconcilable disposition. His place in our profession is unique, for no man hitherto has commanded so wide a reputation, extending beyond both shores of the great Ocean, which he so frequently crossed and recrossed in search of experience, and of increased opportunities of doing good, as well as of gaining recompense and fame.

John Macnaught, a Physician who had practised for twenty-five years in Jamaica, and subsequently for twenty years in Liverpool, died at the house of his son in Kensington, towards the end of last year, at the patriarchal age of ninety years. He had been for forty-one years a non-resident Fellow of this Society.

Born in 1793, he became a Licentiate of the Royal College of Surgeons of Edinburgh, and M.D. of Aberdeen in 1815; and in 1838, F.R.C.P.Ed.

Having gained an excellent reputation in Jamaica, he

returned to England, resumed a course of Medical study in London, and then established himself in Liverpool, where he was known as an able, zealous, and kind practitioner. He was Honorary Physician to the Liverpool School for the Indigent Blind, in which Institution he took a keen interest, and for his services to which he was presented with a piece of plate. As evidence of the esteem in which he was held by his professional brethren, it may be mentioned that in 1865, he was chosen Vice-President, and afterwards President of the Liverpool Medical Institution. The disabilities incident to age, and especially the occurrence of cataract, at length compelled him to retire; but he continued to be a striking example of prolonged vigour, and of the retention of an active and cheerful mind, to the latest days of his existence.

James Manby Gully, the well-known Malvern Hydropathist, was a Fellow of this Society for as many as forty-six years. He died nearly a year since at the age of seventy-six.

Dr. Gully took his degree of M.D. in Edinburgh in 1829. Giving early proof of culture, he published, in 1834, a translation of Tiedemann's 'Physiologie des Menschen,' and in the two following years, a version of Broussais' 'Pathologie Generale,' which appeared in the form of Lectures in the 'London Medical and Surgical Journal,' Vols. vii and viii, 1835-36. Soon after, there appeared in the 'Liverpool Medical Gazette,' his "Remarks on the Rationale of Morbid Symptoms;" and, subsequently to this, he published two essays on the "Symptoms and Treatment of Neuropathy" (1837), and on the "Simple Treatment of Diseases by Expectants and Revulsives" (1842). The tendency therein displayed to some form of Monotherapeutics culminated in his treatise on 'The Water Cure in Chronic Disease,' and in his still more popular works, 'A Guide to Domestic Hydrotherapeia' and 'The Water Cure in Acute Diseases.' His success in this particular line of practice at Malvern, was both rapid and great; but since his retirement, and now after his

decease, he may be said to have left but little mark on the pages of the genuine Medical record.

John Fosse Harding, who was elected into this Society forty-seven years since, appears, for a time, to have been a resident Fellow, for he was a member of the Council in 1858-9. But, latterly, he has lived in the country, for a time at Uckfield, in Sussex, and finally at Hornsey. He was educated at St. Bartholomew's, passed at the Hall and College in 1835, and became a Fellow of the Royal College of Surgeons in 1852.

Adolphus William Leodore Colomiati Meredyth, the first of the *resident* Fellows whose professional career I have to chronicle, belonged to the Society for sixteen years.

Having been educated in Paris, and having served as an Externe at the Hôpital du Midi, and at the Hôpital St. Louis, he was led to volunteer for service with the French army in the East, during the Crimean war. He was Assistant Surgeon to the Barrack Hospital at Scutari, to the Pera Hospital at Constantinople, and to the Osmanli Horse Artillery. He, moreover, aided in founding the Imperial Medical Society at Constantinople.

On returning to France he took the degree of M.D. in Paris, in 1856; and in the following year, he became a Member of the Royal College of Surgeons of England. Being thenceforth established in London, he joined not only this Society but the Medical Society of London, the Harveian Society, and the British Medical Association. He was one of the Physicians to the French Hospital in Lisle Street, and Medical Officer to the French Benevolent Society.

His written works, three in number, were on 'The Duality of Venereal Ulcers' (1866), on 'The Rational Employment of Mercury in the Treatment of Syphilis' (1866), and on 'Hygiene in Relation to Syphilis' (1867).

At the festival held this day last week in aid of the French Hospital, Mr. Crawford, the Secretary, spoke of Dr. Meredyth as one "revered alike for his kindness to

his patients, and his genial disposition towards his colleagues;" while Dr. Vintras, one of those colleagues, speaks of their sorrow at his loss, his devotion to the Hospital, and the deep regrets of his patients.

Philip H. Harper, L.S.A. (1854), M.R.C.S. (1845), and F.R.C.S. by examination (1858), was a Fellow of the Society for seventeen years. He was, for a time, Surgeon to the London Surgical Home for Diseases of Women. He published a paper "On the more frequent Use of the Forceps" ('Obstet. Transac.,' V. i), and another in the 'Brit. Med. Journal' (1860), on "Successful Ovariotomy." He was a devotee of entomological science.

Charles Hilton Fagge was one of those men who attract unusual notice during their lifetime, and whose premature death excites the deepest regret. His early career was so brilliant, and his realised position so conspicuous, that nothing but frail health and a fatal malady interposed between him and the highest professional success. He was a Fellow of the Society for nineteen years, during which period he rendered it both varied and important services. He was a Member of the Library Committee and also a Referee from 1874 to 1879; a Member of a Scientific Committee in the last-named year; on the Council in 1880-81; and, last March, was elected Honorary Librarian, an office which he was obliged to decline owing to ill-health. Considering that Dr. Fagge also contributed seven papers to our 'Transactions,' some special tribute is certainly due to his memory on this occasion.

Dr. Fagge was born in 1838, at Hythe, where his family had practised for two generations. His father, Mr. Charles Fagge, still lives at Hythe; one brother practises at Lutterworth, and another resides alternately at Pau and Arcachon. His mother was a sister of the late Mr. John Hilton, the well-known Surgeon at Guy's Hospital.

As a student at the Great Boro' School, under the auspices of his Uncle, he showed remarkable ability and

power of application. On matriculating at the University of London, he gained, in Honours, the first place in Botany, and the second in Chemistry. At the first M.B. examination, he secured three Exhibitions, viz. in Anatomy, Physiology, and Chemistry, and the Gold Medal in Botany. At the second M.B. examination, he obtained the Scholarship in Comparative Anatomy and Physiology, an Exhibition and Gold Medal in Medicine, a Gold Medal in Surgery, and a second place in Midwifery. Such a wealth of honours had never before, and, I believe, has never since, fallen to any one graduate of that University. Having been chosen a Member of the Committee of Convocation, he took an active part in its proceedings; and, at the time of his death, he was engaged in the responsible work of one of his University's Examiners in Medicine.

Although a Member of the Royal College of Surgeons, Medicine was Dr. Fagge's forte, and distinction in it his ambition. In 1864, he became a Member, and in 1870, a Fellow of the Royal College of Physicians. At Guy's Hospital and Medical School, he passed through many important junior offices, such as that of Demonstrator of Anatomy, Medical Registrar, Lecturer on Hygiene and also on Pathology, and Demonstrator of Cutaneous Diseases. He was elected Assistant Physician to the Hospital in 1867, and after thirteen years of active Clinical work, he was made full Physician in 1880, in succession to Dr. Habershon. He had already held the offices of Physician to the Royal Infirmary for Women and Children, and to the Evelina Hospital for Children.

It is impossible to mention here all Dr Fagge's most numerous and, it may be added, sound contributions to Medical literature and knowledge. To the seven papers which he contributed to our 'Transactions,' between 1852 and 1864, must be added at least twenty others written between 1864 and 1881, most of them for 'Guy's Hospital Reports.' The most noticeable of these were, one on "Poisons," showing the importance of using Physiological tests for their detection, and written many years before

the actual employment of this method in Medico-legal inquiries. Other papers relate to Abdominal diseases and their treatment; to Aneurism, disease of the Heart, and certain rare forms of Cutaneous disease. He translated a work of Hebra's on Skin diseases. He also published a catalogue of the Guy's Museum-models of those diseases, and was the author of the article on "Valvular Disease of the Heart," in 'Reynolds' Cyclopædia of Medicine.' He had accumulated a rich collection of pathological and clinical notes, and had been long engaged in the preparation of a System of Medicine, which it is understood some friendly hand will one day edit for publication.

In all this, we have signs of indefatigable mental energy, proof of great preparedness for teaching, security for winning confidence as a consultant, and a promise of the most substantial success.

The formation of a thoracic aneurism, accompanied by aortic valvular disease, and their inevitable consequences, destroyed these bright prospects. It is said of him, however, in one journal, "Few of those he leaves behind him can be expected to attain the high position to which he seemed so surely and rapidly mounting. It is not only the brilliant physician whose loss to medicine we have to deplore, but, in the case of those who knew him, it is the loss of a true, warm-hearted, and eminently lovable man."[1] In another journal we read—"If an unblemished character, good social standing, proneness to hospitality, troops of friends, a brilliant university career, high professional attainments, and an ardent love for the investigation of scientific problems, could combine to render a man useful to his fellow creatures, and to conduct himself to wealth and fame, they seem to have been so united in the case of Charles Hilton Fagge."[2]

There has just been announced an intention, on the part of his numerous friends, to perpetuate his exceptional fame by some appropriate memorial. He died on the 18th of November last.

[1] 'Lancet,' Dec. 1, 1883. [2] 'Brit. Med. Journ.,' Nov. 24, 1883.

Robert Druitt's vigorous personality and completely accomplished mission suggest a more robust portraiture and a less sad history than those on which I have just had to dwell. He was born at Wimborne, in Dorsetshire, where his father and his father's family had practised for upwards of a hundred years. On his mother's side he was related to the typical Medical family of the Mayos. He died on the 15th of last May, in the sixty-ninth year of his age. He had enjoyed the Fellowship of this Society for thirty years, and he contributed to its 'Transactions' one paper on "Gastrotomy" (Vol. xxxi), and another on "Degeneration of the Placenta" (Vol. xxxvi).

Having received a classical education at the local Grammar School, the benefits of which lasted him through his life, he worked his way deliberately up to the position of a qualified practitioner through the once time-honoured but now out-of-fashion apprenticeship system. Thus, he was articled for four years to Mr. Charles Mayo, of Winchester, came thence, in 1834, to King's College and Middlesex Hospital, and by 1836 and 1837, he duly appeared as a Licentiate of the Society of Apothecaries and a Member of the Royal College of Surgeons.

But the bent of his inclination was to literary work, and his sturdy resolution to occupy his early leisure in this direction, and yet to keep on strictly professional lines, was shown by the publication, in 1839, when he was about twenty-five years old, of the first edition of his ultimately celebrated 'Surgeon's Vade Mecum.' Of this work the industrious and erudite Author prepared, in the following thirty-nine years, ten subsequent editions, the last being issued in 1878. On each occasion, changes, extensions, and improvements, were introduced, and fresh illustrations were inserted; and the total number of copies sold, amounting, it is said, to 40,000, affords unequivocal testimony to Robert Druitt's honest endeavour to render perfect his long-continued and genial task. Perhaps no manual ever manifested greater vitality or enjoyed such uniform and constant favour. Intended at

first for students, it became at length a practitioner's book. Its information was reliable; the views of the various authorities which were quoted in it were precisely set forth; and there were scattered, throughout, in the later editions, many shrewd criticisms and hints, expressed in clear and vivid language. The illustrations, though small in scale, were admirably drawn and engraved, chiefly by the family of the Bagg's. Some of the editions were reprinted, and some were translated, abroad.

The maintenance of his one pet book up to the current state of Surgical knowledge and practice by no means exhausted Druitt's literary force, or filled entirely the intervals of his professional labours. For, during a period of ten years, he was the able and esteemed editor of the 'Medical Times and Gazette.' He published a little volume on 'The use of Chloroform and other means of Producing Insensibility to Pain;' and he was the author of the very learned historical and critical article on "Inflammation," in the last edition of Samuel Cooper's 'Dictionary of Practical Surgery.' Besides this, he wrote numerous official reports as Medical Officer of Health to the Parish of St. George's, Hanover Square; and he found time to formulate his opinions on many sanitary and dietetic subjects, such as "Human Habitations in Relation to Health," "Water Supply," and the use of "Raw Meat," &c. In a famous series of letters, which first appeared in the 'Medical Times and Gazette,' but were afterwards published collectively, he developed his views on the use and value of "Cheap Wines." His minor papers, addresses or letters, were very numerous.

It must be remembered, too, that from the year 1840 to 1872, Druitt was engaged in practice. He became a Fellow of the Royal College of Surgeons in 1845; and, as late as 1874, a Fellow of the Royal College of Physicians also. He was for eight years, President of the Association of Medical Officers of Health; for a short time, Vice-President of the Obstetrical Society; and also an Examiner to the Society of Apothecaries.

Obviously a man of vigorous physique, with a well disciplined and methodically working intellect, he was nevertheless at last compelled to retire from practice, by sufferings of extreme severity. In the midst of these, he continued to work cheerfully on his 'Vade Mecum;' and he received much consolation, in the presentation to him by some of his friends (numbering upwards of 450), of a cup containing £1215, which was offered to him not merely in acknowledgment of his useful labours as an Author, Journalist, and Sanitary Reformer, but by way of furnishing him with a tangible token of their sympathy and regard. There can be no doubt that his work in the world was well and truly done.

Richard Phillips, of Leinster Square, had belonged to this Society for thirty-one years, and served on its Council in 1877. He was educated at Guy's Hospital, and became L.S.A. and M.R.C.S. in 1836, and F.R.C.S. in 1855. In 1857, he published a successful case of "Infantile Hydrocephalus, treated by Elastic Pressure." He was engaged throughout his professional career in general practice, at first in the north of London, but latterly in Bayswater. He enjoyed, in a marked degree, the confidence and esteem of a very large circle of patients and friends.

Nathaniel Montefiore was rather a nominal than an actual or practising member of the Medical profession. He was, however, for thirty-nine years in the ranks of this Society. Educated at Guy's Hospital and Medical School, he obtained the Membership of the College of Surgeons, in 1842, and the Fellowship by election in 1858. He appears to have resided chiefly in London, but frequently in his country house near Southampton. He was extremely well known for the great interest which he manifested in numerous Medical, Scientific, and Charitable Institutions.

Alexander Patrick Stewart, who died last July, nearly seventy years of age, had been for forty-one years a Fellow of this Society. He served on the Council in 1856-7; he

was an Honorary Librarian from 1863 to 1868; and a Vice-President in 1871 and 1872. He was also a Member, and for a time, a Vice-President of the Clinical Society.

Dr. Stewart was born at Bolton Manse, in East Lothian, where his father, the Rev. Dr. Andrew Stewart, a quondam Physician, was the Minister; his mother was the Honourable Margaret, daughter of the tenth Lord Blantyre. Having been sent to Glasgow at the early age of thirteen years, he passed into the Faculty of Arts, and afterwards into that of Medicine; and, having visited the Continent, returned to Glasgow to obtain his M.D. degree in 1838.

In the Infirmary belonging to that city, whilst acting as House-Surgeon, Dr. Stewart was brought into relation with many fever patients; and, as he had already commenced to practise in London in 1839, it was apparently in Glasgow, and probably afterwards in Paris, that he made those clinical observations and records, on which he founded a highly important communication on the "Distinctions between Typhus and Typhoid Fever," which he presented to the Medical Society of Paris in April, 1840, and subsequently wrought into an article, printed in the 'Edinburgh Medical and Surgical Journal' for October of that year.

In this early and noteworthy effort at clinical investigation and analysis, Dr. Stewart really succeeded, according to the late Dr. Murchison, in demonstrating "a distinction between Typhus and Typhoid, founded on a comparison of their origin, proximate causes, course, symptoms and anatomical lesions, including the difference in the eruptions which accompany the two diseases. He pointed out that their characters taken collectively, were, as he said, so marked as 'to defy misconception,' and 'to enable the observer to form with the utmost precision the diagnosis of the disease, and the lesions to be revealed by dissection.'" It has been pointed out that, in Paris, Dr. Stewart would most probably become acquainted with the views of M. Chomel, who, as well as some other observers,

had already suggested a difference between the two fevers; but he was the first to publish such an opinion in this country, and he supported his statements by numerous facts observed and methodised by himself. Partly, perhaps, from his own question, put towards the conclusion of his article, with regard to these fevers, "Are they identical or not?" and partly, from his remark that "all I can ask for is a careful, extensive and minute inquiry,"—but, chiefly, one would believe, from his not having possessed the authority which attaches to age, official station or previously known work, his interesting and valuable contribution to Medicine failed to create the impression it deserved to do. At least, it is known to all present, that it was Sir Wm. Jenner's precise statements and inferences, subsequently published, which cleared away all doubts, and finally determined the general opinion on the subject.

After having practised for several years in London, and having held the office of Physician to the St. Pancras Infirmary, Dr. Stewart was elected, in 1850, Assistant Physician to the Middlesex Hospital, and full Physician in 1855, an office which he resigned in 1866. In the Medical School of that Institution, he lectured first on Materia Medica with Dr. Gordon Latham, and afterwards on Medicine in conjunction with Dr. Seth Thompson and Dr. Goodfellow.

But Dr. Stewart's time and thoughts had already begun to be occupied by other subjects than those connected with clinical study and practice, and by degrees he had become deeply engrossed in them. As far back as 1853, he was a Member of the British Medical Association, and he sat on the Council of its Metropolitan Branch for nearly thirty consecutive years. In this capacity he was assiduous and untiring. During sixteen of these years, in which he was one of the Secretaries, his work was incessant and of the highest utility. He was once nominated President elect of the Metropolitan Branch, and on two subsequent occasions was again solicited to

accept that office, but he always declined on the ground of its burdens and anxieties. He was, however, a Vice-President for three years. As a testimony to his efficient service and estimable private character, the Members of the British Medical Association presented him, in 1875, a sum of money, with a portion of which, handsome breakfast and dinner services were purchased; but the balance, amounting to £400, was generously returned by him to the Association for the purpose of founding a prize for "the Encouragement of Researches into the Origin, Spread, and Prevention of Epidemic Diseases." The first award of "The Stewart Prize" was made, in 1882, in favour of Dr. Vandyke Carter, for his investigations on Spirillum Fever.

Questions relating to Social and Sanitary Reform early attracted Dr. Stewart's attention; for, in 1849, he published an essay on "Sanitary Economics; or, our Medical Charities as they are, and as they ought to be," a serious subject again forcing itself upon our notice at the present time. In 1854, he summarised, in a paper in the 'Medical Times and Gazette,' an account of "The Cholera as it had been observed in the Middlesex Hospital." As to his subsequent literary work, it was no longer clinical, but sanitary. Amongst numerous contributions of that kind made to the 'Transactions of the Social Science Congress,' to the meetings of the Metropolitan Branch of the British Medical Association, and to the Medical periodicals, mention may here be made of one, in conjunction with Mr. Jenkins, on "The Medical Aspects of Sanitary Reform" (1866), and another, so recent as 1882, on "A Home for Convalescents from Scarlet Fever."

A man of the highest principles, and with the strictest adhesion to them, Dr. Stewart was esteemed by all, even by those from whom he differed in opinion, his own being sometimes very warmly expressed. His religious impulses took the practical direction of inaugurating, or assisting in the conduct of Sunday classes for young men, and in missionary services amongst the poor, connected with the

Scotch Church of which he was a member. He did all his work, Medical as well as any other, he said, "much less as a duty than as a privilege." As a Clinical observer, a practical Physician, and a labourer in the field of Medical, Sanitary, and Social improvement, he was, as has been truly said, "an ornament to our Profession."

Thomas William Jones, M.D. of Paris (1834), and M.R.C.S. (1836), was for forty-six years a Fellow of this Society, and held a seat in its Council in 1858. He wrote, probably as his inaugural thesis, "Considerations déduites de l'Anatomie Comparée relatives à differens points d'Ovologie." In London, he was best publicly known as a Physician to the City Dispensary, and to the Provident Clerks' Association. Latterly he retired to Bylochs, Enfield Highway, where he died last May.

William Farr, the *Honorary* Fellow whose death we have had to lament during the last twelve months, died on the 14th April, 1883, in the seventy-sixth year of his age. He had been an Honorary Fellow of this Society for 26 years.

He was born in 1807, at Kenley, in Shropshire, and received his early training in the Grammar School at Shrewsbury. His medical education seems to have been partly carried on in the then so-called University of London, and partly in Paris. Having become qualified in 1832, he acted for a time as House Surgeon to the Infirmary at Shrewsbury; and, thus far, his career was in no way distinguished from that of many accomplished young practitioners of his day.

But there gradually opened for him, in the direction of literary and scientific work, a special path which he henceforth sedulously pursued. He became Editor of the 'Medical Annual' and of the 'British Annals of Medicine,' in which positions he strengthened his unrivalled powers of gleaning and collating facts, and of using them for the purposes of induction.

In 1838, he first entered the Registrar General's Office,

and was soon after appointed Superintendent of the Statistical Department. Herein he continued to labour with extraordinary zeal and success for rather more than forty years. As may be supposed, during this long period of his official life, he received from many quarters marks of consideration and distinction. He was elected F.R.S. in 1855; M.D. of Trinity College, Dublin; D.C.L. of Oxford, in 1857; and a Corresponding Member of the Institute of France, in 1872. He retired from his government post in 1879 with a full pension, and the decoration of Companion of the Bath.

From the date of his appointment in the Registrar General's Office, he was the principal Author of that classical series of Annual Reports, extending from 1837 to 1878, which relate to the births and deaths in different parts of the kingdom. Under his guidance, these documents ripened into full epitomes of the causes of mortality amongst our population, and of the state of health and laws of longevity amongst its various classes. His command of mathematics as an instrument of analysis, his personal study and care in regard to all matters of arrangement, calculation and execution, the logical character of his generalisations, and the usually practical value of his suggestions, were freely acknowledged by all; whilst the occasional introduction of a speculative or even prophetic element into the treatment of his subject, and his often picturesque style, gave to many of these reports a certain charm beyond their avowed utility. The general excellence of his methods has been freely admitted; they have gained a high reputation for our English statistical life-records; and they have supplied models for study and imitation in other countries.

But Dr. William Farr's statistical work extended beyond the compilation of these 'Annual Reports.' Week after week, especially in times of epidemic afflictions, notes and echoes of counsel and warning were issued from his office. He was, moreover, mainly responsible for the general organisation of the Censuses for 1851, 1861, and 1871,

and he prepared the elaborate "Reports" on the movements of the Population therein revealed. He constructed three sets of "Life Tables" founded on the census and mortality records, taking on each occasion a wider and firmer grasp of his complex problems, and solidifying the bases of his calculations for the "Annuity Tables" still in use. He brought his powers to bear upon the elucidation of questions relating to the various kinds of Life Policies, to the proper incidence of an Income Tax, to the due employment of Benevolent Funds, and to the Economics of the Post Office Insurance. He also contributed the article on "Vital Statistics" to MacCulloch's 'Statistics of the British Empire.' In a more strictly Medical direction, he twice reported on Cholera, viz. on the epidemic of 1848-9, and on that of 1866. He aided in determining the precise Nosological terms to be used for the purpose of defining the causes of death in the registration records, and he acted on the Royal Commission for inquiring into the Sanitary Condition of the Army in India.

It is scarcely needful to observe that so eminent a statician as William Farr was an indispensable member of the Statistical Society, which he joined almost at its formation, and on the Council of which his name will be found for forty years. He was a Vice-President of that Society, contributed at least twenty papers to its 'Transactions,' and was appointed its delegate to more than one International Statistical Congress.

"Vital statistics," indeed, were the breath of his mental life, and his name will ever be associated with the history of that department of numerical research. Though short in stature, he had a capacious head, and his mental organisation was evidently adapted for prompt and vigorous, as well as for persistent and solid, work. He also possessed a marked talent for administration and control, for, in his official position and work, he commanded the services of many coadjutors. It was a source of great disappointment to his immediate friends, and especially to those

belonging to the Medical profession, that on the retirement of Major George Graham, with whom he had been so long and so agreeably associated, he was not promoted to the office of Registrar-General. But it was perhaps well for himself, at his age, to be spared further official duty and anxiety. He did not long survive his own consequent retirement, but he must have been gratified and consoled by the warm sympathy and good wishes of his friends, and by the universal acclaim of the rare, honorable, and lasting services which he had rendered to his country.

Having now mentioned the several regretable casualties which have befallen the Fellows of our Society during the last year, I must, in accordance with the promise I made a few weeks since, and doubtless in harmony with your wishes, refer to another loss which we have recently sustained in the sudden and unexpected death of a lamented salaried officer, our late valued Resident Librarian.

Benjamin Robert Wheatley was born at the house of his father, Mr. Benjamin Wheatley, the book auctioneer of Piccadilly, and died in this house, as it may be said, at his post. He therefore lived and died amongst books.

Losing his father in early life, he was left, as the eldest son, the sole support of his stepmother and of a young family. Two years later, his stepmother, to whom he was warmly attached, also died, so that the responsibility of the family henceforth devolved upon him and upon his eldest sister. His two younger brothers have always regarded him as a father, and to him, as they acknowledge, they owe their present position. In his firm devotion to those left to his care, he decided to remain a bachelor, although no one could be more fitted for a domestic life.

After his earlier years, Mr. Wheatley's career was not an eventful one. At the age of seventeen, on leaving King's College School, he assisted in cataloguing for his father the last part of the great Heber Library. This

duty well done seemed to indicate to him his future line of occupation, and from that time to his death he gave up his entire thought and affection to cataloguing and indexing libraries, and to associated bibliographical and literary pursuits.

It was in 1841, that he was first, and temporarily, engaged by this Society to catalogue its Library. Subsequently to this, he performed the same laborious task for between twenty and thirty other libraries, belonging either to public institutions or private possessors. This occupied his time for many years.

In 1855, he was appointed our Assistant Librarian, a title which was recently changed to that of Resident Librarian. In 1856, he completed a new catalogue of our Library, which was then printed; and he prepared and saw through the press our present printed catalogue in 1879. He also compiled for us two indexes of subjects, the latter one constituting the most complete extant English guide to Medical bibliography. Since 1879, he has also produced the supplements to our catalogue.

In all his Library work, Mr. Wheatley was from the first most original, and introduced novelties both of general plan and of detail great and small. His system has been admired and imitated, and will remain as a useful guide in the future. His work, beyond that done for our Library, is recorded elsewhere.

With us, his personal characteristics were, as acknowledged by the Council, "constancy and fidelity in the discharge of his duty," combined with singular unselfishness as to reward. He was uniformly zealous, attentive, and obliging, in all his relations with the Fellows of this Society. He was charged with certain responsibilities beyond those attaching to our Library; for he was engaged in assisting the two other Societies, which meet in these rooms. All this rather heavy work he did indefatigably and conscientiously,—regarding his labours as a pleasure. His last thoughts, and almost his last words, related to our affairs. He had, indeed, served us with his whole heart,

for nearly thirty years. In a letter which he penned some years since, before his salary was raised, he appeals to the Council to consider, in the event of his death, the position of those dependent upon him; and now, notwithstanding his recently increased resources, the Society will doubtless respond to a wish, that, in memory of his faithful services, some provision should be made towards adding to the comforts of his surviving sister and niece.

In the concluding part of my Address last year, I dwelt on a general fact, which appeared to me to convey a striking lesson, drawn from the story of the lives of those whose careers it had been my duty to portray. It was this, that whatever might have been the conditions affecting them, as regards age, education, position, or wealth, all were characteristically *devoted to work*. I ventured by this allusion to indicate that we, the existing Fellows of the Society, might emulate their example.

But, on quitting this Chair, in which I have been placed by your favour and supported by your kindness, it would be unjust not to look beyond the borders and ranks of our own Institution, and wrong to be so blind to what is passing around us, as to assign to our own Fellows, a monopoly, or even a speciality of industry, and an exclusive claim to be considered as guides in reference to what we may accomplish during our lives, or as patterns for imitation after our deaths.

There are other Medical Societies in this Metropolis, comprising numerous able, energetic, and enthusiastic Members, many of whom are, indeed, likewise associated with us. The Medical Society of London, the oldest of the General Societies, founded in 1773, and antedating by thirty-two years our own Society, which was instituted in 1805, is now exhibiting renewed vigour. The Hunterian, established in 1819, and the Harveian in 1831, occupy special ground, and carry out their objects with complete efficiency. In the Appendix to 'Churchill's Medical Directory' for 1846 (the second year of its publication),

these four societies are mentioned. There also existed at that date, the Medico-Psychological Society, established in 1841, and the Royal Medico-Botanical Society which appears now to be extinct. Besides these, there were the Students' and Local Societies, such as the Physical at Guy's (1772), the Middlesex Hospital Society (1774), the Abernethian at St. Bartholomew's (1795), the Westminster Society (1809), King's College Society (1833), and the South London (1845). But, if we consult the 'Medical Directory' for 1883, we find that the Students' and Local Societies have increased from six to fourteen; and that, omitting Societies the objects of which are only remotely allied to Medicine, the general and special Medical Societies in London now number thirteen instead of six, and, moreover, that eight of these have been instituted since the date which I have taken as a starting point, viz. 1845. Of these the Pathological, the first in point of age and strength, was founded in 1846, and now has 690 Members; the Epidemiological was established in 1850; the Odontological in 1856; the Society of Medical Officers of Health, also in 1856; the Obstetrical, which now has 695 Members, in 1858; the Clinical, with its 396 present Members, in 1867; the Dental Surgeons' Association, in 1876; and, finally, the Ophthalmological, in 1880.

Now, it is scarcely possible to estimate the amount of scientific and practical work, which has been, and is being, accomplished by the agency of these various metropolitan societies, whether old or new; but we may freely acknowledge the great energy and enthusiasm of their more active members.

If next we turn our attention to the provinces, we find that there are upwards of fifty Medical Societies, large and small, scattered throughout England and Wales, of which not ten were in existence in the year 1845, and most of which have been founded since the year 1860,—a proof of the ever-developing spirit of inquiry characteristic of our time.

Nor can we fail to be struck with the astonishing growth and present gigantic proportions of the British Medical Association, which, founded in 1832, has now spread out its branches into each of the three divisions of the United Kingdom. Organised for various purposes connected with the welfare of our profession, it has by no means neglected the prosecution of its due share of scientific and practical work. At its numerous annual gatherings, it has its appointed sections devoted to special subjects; it has its communications in each section, the reports of its committees, and its prizes; whilst its doings or Transactions are published in its own Journal. Its local or Branch meetings exhibit a like activity on a smaller scale. Quite recently, too, by means of its Collective Investigation Committee, it has inaugurated a large scheme, with the view of rescuing from oblivion some of that knowledge and experience which, as I hinted at in my last year's address, ordinarily goes down into the grave with its hard-working acquisitors. That the functions of the more important of these societies, whether metropolitan or provincial, are actively discharged, is shown by the reports of their Proceedings, published in their own way, or in the columns of the weekly journals, whose amplified and multiplied pages plainly testify, otherwise, to the manifold industry of our profession at large. New journals also have appeared, published either monthly, quarterly, or at longer intervals, to absorb the ever-increasing amount of original work. The numerous Hospital Reports furnish additional evidence of an enormous expenditure of time and labour. Lastly, it may be noted that whereas in 1846, there were published in Great Britain about 65 original Medical books, in 1881, 1882, and 1883 respectively, the numbers were 108, 119, and 163, besides 56, 58, and 90 new editions.

Time does not permit me to illustrate, as I would wish, the contemporaneous increase of professional activity in Scotland and Ireland; but it has been sufficiently manifest, and requires most ample acknowledgment.

Neither can I do more than allude to the extraordinary development and progress made, during the last thirty or forty years, in the sciences relating to Medicine and Surgery in Germany and France, in Belgium and Holland, in Denmark and Sweden, in Austria and Russia, in Switzerland and Italy, and in the United States. Their great Hospitals and Laboratories, their multitude of Academies and Societies, their long lists of Abhandlungen, Zeitschriften, Archive and Jahrbücher, their Mémoires, Bulletins, Comptes Rendus and Annales, their Acta and Denkschriften, and their teeming individual publications, all bear testimony to this. But I may not omit to notice the numerous general and special Congresses held by our Continental and American brethren, as quite a feature of our epoch, culminating in the International Congress, the great meeting of which, in 1881, we all remember, and which will again assemble in August at Copenhagen. These can only be regarded as great outbursts of otherwise dormant professional energy.

As the mind travels out over this wide field of exertion, and contemplates the operations of the vast hive of modern Medical Investigators, one fails to grasp the total product which they yield; but we may easily perceive how much the various existing Medical Associations must help to stir the ambition and stimulate the zeal of individual agents, in the one common task of advancing Medicine and Surgery. In comparison with the striking scientific advances made and the great practical improvements actually accomplished, during the last thirty or forty years,—on the one hand, the pathological application of the cell-theory, the clearer explanation of the phenomena of inflammation, the new light thrown on nervous diseases, and the discovery of disease germs, and on the other, the splendid triumph over pain by the systematic administration of anæsthetics, the great benefits conferred by antiseptic surgery, the successes of modern ovariotomy, the employment of hypodermic injections, the use of the aspirator, the performance of bloodless operations, and a

host of minor practical improvements—in comparison, I say, with these realised gains, the share taken by this or that Country or this or that Society, in inciting to their initiation or perfection, is of little moment. But yet I must return to our own compact little Body, which though small relatively to the total numbers engaged in the struggle against suffering and death, forms an integral and not ignoble part of the whole.

This Royal Medical and Chirurgical Society has now existed during well nigh eighty years. It has numbered amongst its Fellows all the most distinguished members of our profession in the Metropolis, many Provincial Physicians and Surgeons, and, in its Honorary lists, numerous home and foreign representatives, not only of Medicine and Surgery, but of the sciences related to them. It has held its onward course without serious dissensions from within, and without exciting hostility from without. It has accumulated a splendid library comprising nearly 29,000 volumes, the most valuable circulating medical library in London, perhaps in the world. It has held its fortnightly meetings, bringing men into good fellowship and minds into useful contact. It has published sixty-six volumes of 'Transactions,' consisting entirely of original papers, or of reports from its select committees, and the contents of which are alike remarkable for historic interest and practical value, embracing almost every capital subject in medicine and surgery, and bearing some relation to almost every great achievement in our art.

Although, happily, this Society is so firmly established that its strength and its continued prosperity depend more upon its Members than upon its temporary head, I confess to have felt the great responsibility of the Presidential office; and, when elected, I reflected much on the various modes in which the interests of the Society might best be served. Certain proposals, originating in and adopted by the Council, have, according to our 'Reports,' worked very satisfactorily. Besides these, however, a project, not by any means novel, lingered long in my thoughts,

namely, that of attempting immediately, or at some future time, a combination of certain of the more recently formed metropolitan Medical societies, clustered, as it were, around the Royal Medical and Chirurgical Society as their centre. But the more closely I considered this proposition, the more wise it seemed, at least for the present, to abstain from occupying time and energy by bringing it again before the Council; and the more prudent it appeared to be, to give preference to the plan of pursuing resolutely our own way in the maintenance of our proper position.

In the first place, considering the subject from a general point of view, the previous failures in the attempts at combination were necessarily deterrent as regards another trial; it seemed to me doubtful how far any fresh proposal of the kind should originate with the President; rather it appeared to be more appropriate that it should proceed from the Council itself; further it was open to question whether it should not be suggested by the younger Societies to us, and not by us to them; and lastly, I thought that in any case the best preliminary step towards such a union, if it were to be satisfactory, would be to maintain our own status and reputation, or, if possible, to elevate the one and increase the other.

In the second place, looking more closely into the subject on its merits, it seemed that though the contemplated union might satisfy the desire for centralization and furnish the means of securing certain economies, it might have its difficulties and its disadvantages. Each one of these younger Societies (for there is, I presume, no question as to the three elder ones) obviously arose from a felt want; its suggestion was quickly responded to, its establishment forthwith followed. By each, much new and admirable work has been achieved, in some instances, especially in that of the Pathological Society, as remarkable for its quality as for its amount. Nor should this be a matter of surprise; for it is usually held that division of labour is not only a sign, but a need and a cause of advancement. As

the circle of knowledge is progressively widened, its marginal explorers stand further and further apart from one another, the horizon of investigation is seen by them from more and more widely separated points of view, and room is made for additional intermediate workers. It may well be doubted indeed, whether if, in place of enjoying the freedom and stimulus to action resulting from an independent organisation, these Societies had been incorporated with our own as sections of a larger scientific body, so much good work would have been accomplished in the past; and it is by no means certain that under similar conditions of union, more would be done in the future.

A recent example of such a combination seems, however, full of promise, and may, some day, encourage a further attempt amongst ourselves. In Dublin, four already established Societies, the Medical, Surgical, Obstetrical, and Pathological, have been merged in the "Academy of Medicine in Ireland," in which they are now represented by four corresponding Sections, to which two subsections have been added, one for Anatomy and Physiology, and one in relation to Public Health. The details of the plan have been carefully considered, the rules are simple, and, as a beginning, the experiment is highly satisfactory. But the conditions of the problem in Dublin are very different from those which exist in London. The population of Dublin and its immediate vicinity is about 300,000; that of London and its suburbs above 4,000,000. In Dublin and its neighbourhood, there are less than 600 Medical Practitioners of all ranks and titles; in the total Metropolitan area, there are about 4000. In the voting for or against the formation of the "Academy of Medicine in Ireland," 192 suffrages were cast in favour of, and 20 against the proposal, making a total of 212 individuals concerned in the proceeding; whilst the Academy itself numbers at the present moment 204 Fellows and 24 Members,—there being two classes of Associates. On the other hand, in London, it would be necessary to consider the wishes of between 600 and 700

individuals, who would be interested in any new arrangements.

It must not be forgotten, moreover, that the opportunities and materials for observation and study—whether for Pathological or Clinical, Medical, Surgical, or even Obstetrical purposes—are widely different in the two cities. In Dublin, there are now twenty-eight Institutions for the reception of the Sick, including Hospitals, Infirmaries, and Dispensaries; whilst in London, besides the eleven large Hospitals, there are upwards of 170 smaller Charitable Medical Institutions. So rapidly have they increased, that in the year 1846, a date I have previously referred to, only sixty-three of these were in existence; and by far the larger number of the 110, since added, have been instituted since 1860. This is altogether independent of Poor Law, Union, or Parochial Establishments for the Sick Poor. That many of these numerous centres of medical observation yield little material, of novelty or of interest, is very true; that none of them are, at present, utilized for the purposes of Clinical teaching or practice, as they might and ought to be, has been long urged. But it is undoubtedly from them, viewed as a whole, that rich stores of morbid specimens are now obtained for microscopic investigation, and, in them, that many careful Clinical studies are conducted. As President of the Royal College of Surgeons, which is deeply interested in perfecting its noble Pathological Collection, I may be pardoned for reminding those who may hear or read this Address, and who are in the habit of resorting to these sources of material for the advance of knowledge, how much they may be able to assist in enriching the College Museum.

As the retiring President of this Society, I point to these numerous Institutions as constituting a vast field of research, filled with so overwhelming an amount of the undetermined and the unknown, that their existence supplies a powerful reason for avoiding any step which might endanger the continuance of the active energy, which

happily distinguishes the separate and somewhat rival organisations which are now employed in their utilisation.

I believe that we may bequeath the amalgamation of the existing Metropolitan Societies as a legacy for the future. I do not say it should not, or will not, come to pass; but, for the moment, it seems to me that their free and independent action, and their separate status, is so much more in accord with the immense work to be accomplished, and so much more equal to its achievement, that I am satisfied, or even more than satisfied, with that independence, and I would here most heartily wish them all possible success. So certain are partial acquisitions to lead to a general gain, that I believe this Society, which may claim to be *general* and *non-exclusive* in the highest and widest sense, will ultimately profit in its own sphere of utility by the auxiliary exertions of the others; and in the fervent hope and firm belief that this will prove to be so, I now resign my office, feeling the utmost confidence in the future progress and continued prosperity of this distinguished scientific Body.

CEREBRAL LOCALISATION.

A REVIEW AND FORECAST.

BEING THE

MARSHALL HALL PRIZE ORATION.

BY

DAVID FERRIER, M.D., LL.D., F.R.S.

(Received October 23rd—Read October 23rd, 1883.)

Mr. President and Fellows of the Royal Medical and Chirurgical Society,—I have in the first place to express to you my grateful appreciation of the great honour you have done me in awarding me the Marshall Hall prize of this year, and of the further high privilege you have accorded me, in asking me to address you on this occasion.

To be the recipient of a prize associated with the name of Marshall Hall, and for work done in that department of medical science which he himself so adorned and enriched, is a distinction of which I am indeed proud. I know of no name in the ranks of scientific medical inquiry in this or any other country which stands higher than that of Marshall Hall, and no work which has done more to advance the physiology and pathology of the nervous system, and not this only, but medicine and surgery in general, than his investigations into the nature and conditions of reflex action. This he was undoubtedly the first to formulate and expound, and clear from the vagueness

and confusion which prevailed before him. The importance of his researches can scarcely be over-estimated. If it were possible to eliminate from modern medicine all that relates to reflex action, its bearings and applications, we should practically de-rationalise the better half of pathology and therapeutics. Few scientific inquirers have been more keen to perceive the practical bearing of their own researches than Marshall Hall himself; and most of the principles which guide us in the recognition and treatment of diseases and symptoms dependent on reflex irritation, were first clearly laid down by him.

Not long prior to the time when Marshall Hall was pursuing his investigations on the spinal cord and medulla oblongata, Flourens was engaged on his ever memorable researches on the physiology of the cerebral hemispheres. And Flourens also, to whom he dedicated several of his memoirs, was almost the first to perceive and recognise the value of the work done by Marshall Hall which many of his countrymen endeavoured vainly to detract from and depreciate.

If, at the distance of fifty odd years, we compare the relative stability of the work done by these great men, that of Marshall Hall on the spinal cord, and that of Flourens on the cerebral hemispheres, we find that the doctrines enunciated by Marshall Hall, modified perhaps as to detail, and further extended by the numerous researches of recent years, are in all essential points those which still prevail, and show no signs of failing; while those of Flourens which have also exercised an enormous influence on clinical medicine and pathology,—What shall we say of them?

In the brief sketch which I purpose giving you of the present position and probable future of this question, I fear that however much I may try to avoid it, I shall appear more in the character of the advocate than the impartial judge. But I am fully conscious that I am before a highly critical audience who will not allow any bias of mine to warp their own just judgment.

The views of Flourens are familiar to you all. They seemed to the scientific world at once in accordance with the facts of experiments on animals—at least of the lower orders—and in harmony with the prevalent metaphysical conceptions as to the unity and indivisibility of mind. Like the mind itself, said Flourens, the organ of mind was also one and indivisible; there being no differentiation of function, but each and every part possessed of the potentialities, and capable of exercising every function, pertaining to the whole. These doctrines speedily met with general acceptance among physiologists, but there were a few keen clinical observers, Bouillaud, Andral, and others, who saw in the phenomena of cerebral disease facts which appeared wholly inexplicable except on the hypothesis of a differentiation of function in the cerebral hemispheres; such for instance as the occurrence of limited paralyses in connection with limited lesions. But unable, on the other hand, to account for the facts of experiments on animals on the principles of localisation which the clinical data seemed to demand, they wisely suspended their judgment, neither denying the facts of experiment nor doubting their clinical observations, believing that the apparent discrepancies between human pathology and experimental physiology would one day be cleared up and dispelled.

This was eminently the attitude of my distinguished predecessor in the honorable position I now hold. Hughlings Jackson, neither contesting nor trammelled by the doctrines of the "school-physiology," saw in the occurrence of limited and unilateral muscular spasms in connection with certain cortical lesions, phenomena which to him signified irritation or discharge of grey matter directly related to muscular movements; and with a keen and prophetic insight, which far outran the slow march of verified fact, he evolved many brilliant and fruitful conceptions as to the anatomical substrata of the organ of mind.

There were, however, many other clinical facts, particularly those relating to loss of speech in connection with

lesions of the left hemisphere, which formed the subject of so much lively debate and discussion twenty years ago, which remained equally obscure and mysterious, whether on the hypothesis of localisation or the reverse. It is not going too far to affirm that up to the time when the researches of Fritsch and Hitzig inaugurated a new era in cerebral physiology, general doubt, difficulty and confusion prevailed. It would even now be premature to claim that all this has been dispelled, but we may say truly that the questions in dispute have assumed definite and manageable shape, and that every day the dark atmosphere is being cleared.

I will not weary you with an account of the controversies which arose as to the true significance and interpretation of the results which followed the application of electrical irritation to different regions of the cerebral hemispheres.

On this, as well as regards the consequences of destructive lesions of the cortex the most divergent views continued to be entertained and expressed.

The fundamental question "Localisation or no localisation?" was brought to a crisis at the meeting of the International Medical Congress here in 1881.

You are all aware, many of you doubtless heard and saw, that Prof. Goltz, the chief of the few remaining champions of the Flourentian system, brought with him from Strasburg a dog in which he had, long previously, at intervals destroyed a large extent of the cortex of both hemispheres, and which he exhibited before the physiological world as a practical refutation of the theory of cerebral localisation. He enunciated the following theses.

1. The cerebral cortex is the seat of the higher intellectual functions. Removal of large portions of both hemispheres degrades the intelligence.

2. It is impossible by any localised cerebral lesion to cause paralysis of any muscle. The animal operated on, retains volitional control over all its muscles.

3. It is also impossible by any localised cortical lesion to cause permanent loss of any sense. The animal retains

all its sensory faculties. After removal, however, of large portions of the cortex defective perception is induced.

4. Animals in which the parietal regions have been destroyed are permanently awkward in their movements, and defective as regards tactile sensibility. Animals in which the occipital lobes have been destroyed are, as a rule, more demented than those in which only the parietal regions have been destroyed.[1]

He pointed to his dog as an illustration of the truth of these propositions; awkward in all its movements, especially those of the hind legs, but paralysed as to none; defective in all its faculties of sense, and sensory perception, but neither blind, nor deaf, nor otherwise totally deficient.

Some exceptions were taken by my colleague, Prof. Yeo, and others as to the exact accuracy of Prof. Goltz's description of the extent to which the respective motor and sensory faculties were impaired, but I pass over these as not essential to the matter in hand.

When the brain of the dog was examined by a committee appointed by the section, it was found that the lesions, though extensive in both hemispheres, were less than Prof. Goltz had imagined, and did not implicate the *whole* either of the so-called motor regions, or of the regions of special sense as defined by his opponents.

Over against Prof. Goltz's dog were set two monkeys, in the one of which the greater portion of the region in the left hemisphere called motor had been destroyed many months previously. The animal had remained hemiplegic on the right side from the date of the operation, and at the date of exhibition presented the typical features (viz. the semiflexed arm, and rigidly clenched fingers) of hemiplegia with late rigidity as seen in man. The hemiplegia was seen and admitted by all, and the permanency of the result was sufficiently plain to all conversant with the incurable nature of hemiplegia with late rigidity in human patients. It was afterwards proved, by a committee of investigation, that the lesion in this case affected only the

[1] 'Trans. Internat. Med. Congress,' 1881, vol. i, p. 228.

cortical matter and subjacent medullary fibres in the region where it was stated to be; and the fact of descending degeneration of the pyramidal tracts of the right side of the spinal cord was also established at a later date.

With the exception of the motor paralysis of the right side, the animal was in all other respects perfectly well; full of intelligence, and acute in all its sensory faculties and powers of perception.

The other animal in which the superior temporo-sphenoidal convolution had some time previously been destroyed on both sides, presented a marked contrast to the first. There was no question as to this animal's motor powers, for it bounded about and climbed hand over hand with the greatest agility and vivacity; nor was there any question as to its sensory faculties except one, viz. hearing, and in this respect it was admitted to be totally deficient; for as was remarked by one present, it was the only animal in the room that was not startled by the explosion which I made.

In this condition of total deafness, with retention of all its other faculties both sensory and motor, it had been since the operation and continued so till its death a long period subsequently.

The brain of this animal was exhibited at a meeting of the Physiological Society, and will be described in another place. Suffice it to say that the lesions were confined to the cortical and subjacent medullary fibres, in the regions I have indicated.

It is at once obvious that the second and third of the propositions enunciated by Prof. Goltz, and it was so admitted by himself, are demonstrably erroneous as applied to monkeys, and that in their case the localisation of function was incontestably proved.

To some, however, it seemed as though these demonstrations instead of definitely settling the question of cerebral localisation, had made confusion more confounded. A word or two, therefore, on the lessons taught on this occasion, and on the conclusions to which they logically lead.

Localisation of function being admittedly established in the case of one vertebrate animal; then, given facts of experiment on another animal of the same type seemingly at variance with this law, there is no alternative between either denying the universality of the law, or holding that the facts though seemingly at variance are in reality in perfect harmony with it.

I cannot conceive it possible that in the present state of biological science any one will be found to maintain that animals constructed on the same anatomical type can differ so fundamentally in their physiological constitution as would be implied in the first alternative. We may assume, therefore, that the whole question turns on how, on the principle of localisation, we are to account for such striking differences as are observable between the effects of destruction of the cerebral hemispheres in the different orders of animals.

It would be difficult on cursory examination to say whether a frog or pigeon had had its cerebral hemispheres removed or not. For they each maintain their normal attitude, and the one will hop and the other walk or fly and otherwise respond to sensory stimuli in a manner little, if at all, differing from the normal. The universal powerlessness, however, of even a dog, and much more so of the higher vertebrates after a similar operation would be very apparent.

These facts, admitted by all, clearly demonstrate the existence of great differences in degree among the different orders of animals, in respect to the part which the cerebral hemispheres play in the ordinary modes of activity, and response to various forms of external stimulation.

If it is difficult in some animals to detect the results of removal of the *whole* of the cerebral hemispheres, how much more difficult must it necessarily be to determine the result of removal of only a portion. No one would think of denying a definite function to the spleen, though it is confessedly difficult to determine the results of its extirpation.

The neglect of the most obvious lessons of comparative

physiology has been the cause of much useless and barren controversy :—facts being urged in opposition to other facts between which no antagonism really exists. If to these considerations, founded on comparative physiological research, we add that unless the *whole* of a given cortical centre be destroyed, the portion remaining may suffice for the continuation, in some degree at least, of the function allotted to it, we may arrive at a completely satisfactory explanation of the condition of Prof. Goltz's dog in thorough harmony with the fundamental principle of cerebral localisation.

I have purposely excluded the evidence of clinical medicine in reference to this question; for the exact solution of problems in physiology by the fortuitous experiments of disease in man is always difficult, and nowhere more so than in the domain of cerebral function. The phenomena of cerebral disease are so complex, the factors at work are so indeterminate, that they seldom admit of exact analysis and computation. But though of themselves clinical observations may be unable to carry rigid conclusions as to cause and effect, all physiological generalisations otherwise founded must square with the facts of clinical observation before they can be admitted as an integral part of the institutes of medicine. And on many points, particularly where subjectivity is concerned, observations on man constitute the only reliable means of interpretation of vital manifestations.

Apart from some differences in matters of detail which do not affect the main principle, localisation of cerebral function is accepted by every recent writer on cerebral disease, as the only possible explanation of the phenomena. Mere numbers and authority, however, will not stand for evidence, and I do not quote them as such. But, as it would be impossible for me to set forth the evidence here, I can only refer inquirers to what I have elsewhere written on the subject, and to the facts contained in the more recent publications of Charcot et Pitres, de Boyer, Grasset, Nothnagel, Exner, and Ross, as well as to the

cases, such as those recently recorded by Sharkey, which are constantly appearing in every medical journal.

Assuming therefore that the principle of cerebral localisation, has become established in physiology and clinical medicine, the ground is cleared for the consideration of further questions of great scientific and practical importance, on which opinion is not unanimous, and on which therefore further research is desirable. The first is, is the localisation of centres a matter of indifference or accident; or is it dependant on structural peculiarities and connections which render each centre as distinct from others, as, *e.g.*, one limb from another, or the organ of vision from the organ of hearing? Or, to put it in the form of a simile, is the brain crust divided into fields each of which though yielding one kind of produce *usually*, may if need be, yield any other; or into fields which yield only one kind of produce, and *can* yield no other; or, according to a still third supposition, is it divided into fields, each of which yields *most* of one kind of produce, but also more or less of every sort.

Each of these three suppositions has its advocates and defenders. If the localisation is merely accidental or indifferent, perhaps the function of a part lost, may be taken on by some or other of those remaining.

If it is structural, this would be as impossible as that the organ of hearing should perform the functions of the organ of vision, or that the nervous apparatus which moves the leg should also move the tongue, or act as the centre of taste.

The question is one which ought to admit of definitive settlement by properly directed research.

The indifferent theory has been adopted by many, under the name of "functional compensation," as affording the most satisfactory explanation of the apparent recovery after destruction of the cortical centres of motion in certain of the lower animals.

If it should appear, however, that in monkeys and still higher animals, it is possible by total extirpation of a

given centre, to cause complete and permanent annihilation of a given faculty of sense or motion, all others remaining intact, it is obvious that the hypothesis of functional compensation is uncalled for.

Those who consider this established, explain the apparent recovery in the lower animals, on the principles already laid down respecting the differences in degree in which the motor faculties, and powers of response to sensory stimuli, are affected by destruction of the cerebral hemispheres in different orders of animals. The so-called functional compensation of the hemispheres is in reality no compensation at all, but only a manifestation of the activity of lower centres.

In addition to the facts of experimental physiology demonstrating the possibility of entirely and permanently annihilating a given function or faculty, others are adduced in favour of a rigid structural localisation of centres.

Though the grey matter of the cortex appears similar throughout, and composed of elements common to every region more or less, yet recent histological researches, particularly those of Bevan Lewis, have succeeded in determining the existence of numerous peculiarities in structural arrangement and collocation in different regions of the cerebral cortex.

The significance of these structural peculiarities is by no means apparent, but that they exist and have a meaning, which may one day become clear, there is no reason to doubt.

Next, and perhaps of more intelligible signification than these histological characters, are the facts, now indubitably established, both by physiological experiment and human pathology, that when certain regions of the cortex, viz. those termed motor, are destroyed, *and no others*, secondary degeneration ensues in the pyramidal tracts, down the whole length of the spinal cord.

The inference drawn from these data is, that just as the motor nerves are connected with the anterior horns of the spinal cord, so the pyramidal tracts of the spinal cord are

connected with certain regions of the cortex cerebri, showing therefore a fundamental difference as regards anatomical connections between one region of the cortex and another.

It is argued also that if the pyramidal tracts had other connections, destruction of other regions than the motor would induce secondary degeneration also,—which is not the case; and that these connections if they existed should prevent the atrophy and degeneration which occur on destruction of the motor area.

As regards regions related to the organs of sense, it is claimed as an established fact, that the destruction of a certain cortical region on both sides, causes complete and permanent loss of vision, followed in due course by atrophy of the optic discs.

This is relied on as proving that this region is anatomically the cerebral expansion of the optic tracts; and that the complete abolition of vision, and subsequent atrophy of the optic nerves, show that this region is alone the centre of vision; and that if the optic nerve has connections with any other region or regions, these are unable to form the substrata of the visual sense, or to prevent wasting of the optic nerve.

No similar observations have as yet been made on the trophic relations, if any, between particular cortical areas and other organs of special sense.

Some cases have however been published in favour of the occurrence of atrophy of certain cortical regions, as the result of long standing deprivation or congenital absence of some limb or organ of sense. Many of them have, however, an extremely slender foundation, and much is required in the way of future research before conclusions can be safely founded on them.

But if succeeding research should establish these facts to be free from doubt and uncertainty, and if all the statements should be substantiated in reference to the permanence of the effects of cortical lesions, and the consecutive degeneration of certain tracts and organs, the question of in-

different or rigid structural localisation will have been definitively settled.

After the fundamental principle of localisation itself however, all other points involved in this, whatever may be their scientific interest, sink in importance, as regards practical aims, in comparison with those relating to the exact delimitation of the respective cortical centres. Here we meet with considerable diversity of opinion among those who have experimentally and clinically investigated the subject. This has been made use of by some, and very illogically, as an argument against localisation altogether. It might be alleged with just as much reason that any differences of opinion among the Fellows of this Society on questions brought before them, are a sign of instability on the part of the Society itself.

I should require a treatise, rather than the limits of a brief sketch, to place before you the various facts and arguments on each head, and I should have to import many things of my own as yet unpublished, for which this is neither the time nor place.

I content myself, therefore, with merely indicating the points in which there is more or less harmony, or the reverse.

The convolutions bounding the fissure of Rolando are, without exception, regarded as having a special relation to the motor powers. Whether motor disorders are invariably caused by lesions in this region is a point of capital importance. If clinical research should establish the existence of an unexceptionable case of total destruction of this so-called motor region without motor paralysis, such a case will do more towards demolishing the whole theory of the existence of motor centres in the human brain than the many hundreds of cases adduced in support of it. But naturally, considering the harmony which otherwise exists between the positive clinical instances and the established data of experimental physiology, we have a right to demand something more than mere vague assertion or crude investigation before a negative instance can be admitted as

proved. None such has as yet been put on record. All the doubtful cases, extremely few in number, bear only on the precise limits of the respective centres, and not on the general question of the existence or not of a special motor area.

The centres of hearing have been shown before the physiological world to be in the superior-temporo-sphenoidal convolutions, and numerous clinical observations have been put on record favoring the existence of a causal relationship between lesions of this region in the left hemisphere, and the occurrence of the defect termed "word deafness," —a defect in the re-presentative functions of the auditory centres. Nothing, however, comparable to the absolute deafness, presentative and re-presentative, seen in monkeys from bilateral destruction of these regions, has as yet been observed in man.

The centres of vision are still the subject of some differences of opinion; but these have reference not so much to the general position as to the exact delimitation of the anterior boundary, and to the relations of each centre to the central and peripheral portions of the retina respectively. Clinical observations are being published in considerable number demonstrating the occurrence of isolated affection of vision in connection with cortical and subcortical lesions in the occipito-angular region; and the differences between the published views on the exact relations of the centres and organs of vision, are in process of being settled.

The centres of smell and taste are less definitely determined, owing to unavoidable difficulties in experimentation; but the greatest differences of all obtain in reference to the localisation of the centres of common and muscular sensibility. A considerable number of physiologists place them in the so-called motor region; while I have all along held, and hold, both on experimental and clinical grounds, that the centres of common, including muscular, sensibility, are anatomically entirely distinct from those of motion, and situated in a special cortical region.

It is of the utmost importance that this question should be settled, for it involves vital issues in the regional diagnosis and differentiation of cortical from other cerebral lesions. In this respect clinical observation—careful and accurate work—has a transcendent value; and there is every reason to believe that this, in combination with the more precise experimental method, will at no distant date succeed in solving outstanding difficulties, securing general harmony of opinion, and placing cerebral localisation and the regional diagnosis of cerebral disease on a firm and unassailable foundation. In no department of medical science is more activity being manifested; and there is no field more inviting or more likely amply to repay cultivation.

The following words of Marshall Hall are perhaps more pertinent now, than they were to his own time: "In every point of view there is much to be done. Every encouragement should be given to the diligent and devoted investigator; every obstacle, every kind of injustice, every source of disgust and of indignation, should, for the sake of science, for the honour of our institutions, be removed. The physician, who devotes himself to investigation, especially, makes a thousand sacrifices; his path requires cheering, and should not, as it need not, be unjustly obstructed or beset with thorns."[1]

Though the value of scientific investigation is by no means to be measured by its practical utility, yet the value of a scientific fact or principle is enhanced in the eyes of all mankind when it is useful as well as true.

Tested by the standard of practical utility, what, it is asked, has cerebral localisation done, or is it likely to do, towards a more successful treatment of cerebral disease than we yet can boast of?

Up to the present I think that, with a few, though significant exceptions, the benefits of the scientific doctrine of cerebral localisation have been absorbed, like so much

[1] 'New Memoir on the Nervous System:—On the True Spinal Marrow,' p. 94.

latent heat, by medical science itself, as distinct from medical or surgical practice.

It has been a lamp to lighten the path of the clinicist through darkness almost chaotic; it has sharpened clinical vision, so that now many things are clearly seen which were formerly supposed not to exist; it has given a precision to clinical and pathological descriptions which will be searched for in vain in the older records of cerebral disease; it has cleared our conceptions as to the significance of numerous symptoms, and rationalised many purely empirical generalisations, and is every day bringing us nearer to that which Virchow has termed the goal of modern medicine; viz. the localisation of disease.

But when this has been reached as regards cerebral disease, when we are able to determine the exact nature and position of the materies morbi; is it at all likely that we shall stop here? This would be very improbable, even though we should as yet have none, or only the most vague notions in what direction practice might be influenced.

But there are already signs that we are within measurable distance of the successful treatment by surgery of some of the most distressing and otherwise hopeless forms of intracranial disease, which will vie with the splendid achievements of abdominal surgery. What can be more terrible than the agonising headache, the torturing sickness, the racking convulsions, the loss of sight, the progressive paralysis and mental infirmity, and miserable death from a cerebral tumour, which we daily see and foresee, and are powerless to avert by any means known to our art? It is natural that physicians should hesitate to advise surgical operations on the brain, and that surgeons should stay their hands until the principles of diagnosis of reachable disease should have become established with as near an approach to certainty as is possible, where all is hidden from the eye and hand.

But granting this, is there any reason why a surgeon should shrink from opening the cranial cavity, who fear-

lessly exposes the abdominal viscera? The peritoneum was until a very recent date held sacred and inviolable. The dura mater and brain are much in the same position now.

But after what I have seen of the unfailing safety, the freedom from all untoward results as regards health and life, with which the most formidable, and repeated operations can be performed on the brain and its coverings, under stringent antiseptic precautions,—and these on animals of the most delicate, almost human, organisation, —I cannot but believe that similar results are capable of being achieved on man himself.

Apart from secondary inflammation and its consequences, which can be absolutely prevented, there is no risk to life from even extensive destruction of the cerebral hemispheres. It is true that in attempts to remove tumours, or locally treat other forms of disease, we may injure or destroy healthy portions of brain tissue. And what will follow?

Not necessarily mental disorders or appreciable mental defect. With the exception of certain functions arrogated by the left hemisphere, we have for mental purposes practically two brains; and diseases are not always on the left side. The records, and our daily experience of disease and injury of the brain, show that considerable portions of brain substance may be destroyed without great danger to mental stability. But paralysis may ensue, more or less extensive, or defects in special sense, according to the position and amount of the lesion. Such risks undoubtedly exist, as well as others incident to the operation itself, as also to all surgical operations.

But the choice is not between this and any other mode of treatment, but between running these risks and certain death.

And I have no doubt that there are many who, if they had the choice, would go through life, if not enjoying it, halt or hemiopic, rather than perish miserably after, it may be, years of incessant suffering.

Ne sutor ultra crepidam! I think I hear remarked.

I applaud the sentiment, but as in this matter I have had the experience of the surgeon, as well as of the physiologist and physician, I have thought that I might venture on these suggestions without appearing to go very far beyond my "last."

CASE

OF

SPONTANEOUS INGUINAL ANEURISM

IN A BOY AGED TWELVE YEARS;

FOR WHICH THE EXTERNAL ILIAC ARTERY WAS TIED.

WITH

A TABLE OF ALL THE OTHER RECORDED CASES OF EXTERNAL ANEURISM IN PERSONS UNDER TWENTY YEARS OF AGE.

BY

ROBERT WILLIAM PARKER, M.R.C.S. Eng.,

SURGEON TO THE EAST LONDON HOSPITAL FOR CHILDREN.

(Received October 5th—Read November 13th, 1883.)

The rarity with which external aneurisms are met with in young persons induces me to lay the present case before the Society.

Frederick L—, æt. 12 years and seven months, a German by birth, was admitted into the East London Hospital for Children on October 31st, 1882.

Previous history.—The patient's mother having died five or six years previously, no very reliable history could be obtained. As far as could be ascertained there was no evidence of rheumatism nor of syphilis. He had began to suffer from hip-joint disease on the right side when between two and three years old. This was said to have been caused by an injury. Owing to this disease he had

used a crutch, and though going regularly to school had never engaged in rough play like other boys.

Present illness.—This began suddenly. On October 1st he went to bed in his usual health and was roused up from sleep in the middle of the night by a "cutting" pain in the left groin running down the thigh towards the knee; however, he got up next morning and went to school as usual. In a "day or two" the boy noticed a "lump" in his groin; the pain continued and was so aggravated by walking that he gave up going to school. The lump was not seen by a doctor until two days before his admission to the hospital.

Condition on admission.—The boy is very pale and emaciated. His fingers and toes are slightly clubbed. In the left groin, just below Poupart's ligament, there is a swelling in the course of the femoral artery, which pulsates. This swelling is somewhat square in shape, measuring about two inches from side to side. The pulsation is most marked along the outer border of this swelling, where it passes beneath the Sartorius muscle; it is so forcible that a pulsatile movement is apparent on the outer surface of the thigh just above the trochanter. This pulsation is quite arrested when pressure is made on the external iliac artery. A purring vibration is felt in the arteries below the aneurism, as low down as the dorsalis pedis. The pulsation below the aneurism is much weaker than on the opposite side. The superficial vessels of the leg are not enlarged, neither is there any œdema. The thigh can be fully extended, but some pain is felt in the groin on making extension. Slight (reflected) pain is also present on the inner aspect of the knee. His right femur is firmly anchylosed to the pelvis at an angle of 115°. There is no pain or tenderness whatever in this limb; but the purring vibration described in the left leg is felt in all the arteries of the right leg down to the foot. This is best appreciated by grasping the limb with the hand; it suggests the sensation of small hard bodies circulating with the blood. The pulsation in the articular arteries about

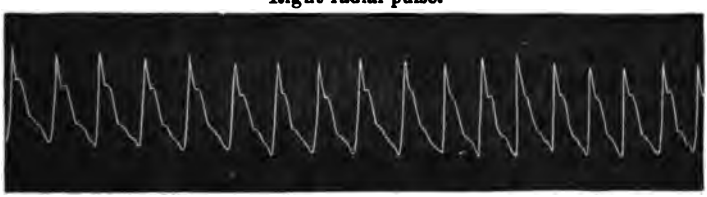
the knee is very visible and striking; these arteries all appear to be enlarged.

On inspecting the chest, which is of a very rickety type, a strong, heaving pulsation is noticed about the inner ends of the clavicles, while the visible cardiac pulsation extends for some distance beyond the left nipple. On percussion, cardiac dulness is found not to extend beyond the right border of the sternum; it is absolute as far as the mid-line of the sternum, and for about half an inch outside the nipple line; it commences above in the second space and reaches down to the sixth. On auscultation a loud constrictive and a regurgitant aortic murmur are audible at the base. There is a soft mitral murmur at the apex, which is audible also at the angle of the scapula. The lungs do not present any signs worthy of record; breath sounds are weak.

The right radial pulse is markedly stronger than the left, the reason for which is not very obvious; both beat 112 per minute, and are of the "water hammer" type.

The following tracings were obtained; 4 oz. pressure used in each case.

Right radial pulse.



Left radial pulse.

The fundus of the eye is found to be quite normal. The urine is healthy.

As the boy had been badly fed for a long time past it was decided not to operate at once; he was kept quiet in bed, and put upon a generous diet.

On November 10th, eleven days after his admission, I ligatured the external iliac artery under a carbolic spray, using for the ligature Chinese twist-silk well soaked in 5 per cent. carbolic acid solution. There was no obvious disease in the artery at the spot tied. The operation presented no difficulty whatever. The limb was subsequently enveloped in cotton wool. At 8 p.m. the circulation in the limb was good and the toes of the left foot were almost as warm as those of the right. The wound healed by the first intention, except at the spot where the drainage-tube emerged.

On November 18th a raised erythematous blush was noticed on his left cheek, and there was some elevation in his temperature; a few days later this redness had assumed an erysipelatoid character, and spread across the nose to the right side of the face; his temperature rose to 102° F.

On December 4th, at 10 p.m., he vomited some blood, and a motion which he passed about the same time also contained blood. It was then found that hæmorrhage was going on from the posterior nares; this continued more or less all night though various measures were ineffectually tried to arrest it.

On December 5th, at 9 a.m., he was blanched; his temperature was 97° F., and his pulse hardly perceptible. On examining the pharynx a slight trickling of blood could still be seen. At 6.30 p.m. he vomited profusely, after which he gave two or three gasps and died.

The autopsy was made twenty-four hours after death. In the head all the arteries were carefully examined; no lesion, obstructive or otherwise, was discovered. The membranes and substance of the brain were healthy; there was no effusion into the ventricles. The lungs were everywhere adherent to the chest walls; they were rather œdematous, but presented no trace of pneumonia nor of infarcts. The liver and spleen, stomach and intestines, and the kidneys were all quite healthy. The right hip-joint was firmly anchylosed to the pelvis; within its capsule was

some inspissated caseous matter. The heart was removed together with the aorta, and its terminal branches as far as the popliteal spaces. Beyond the aneurism itself no other abnormality of any kind was detected. Portions of the arterial walls were subsequently examined microscopically with a like negative result.

The ventricles were much hypertrophied, the left one being quite half an inch thick. The mitral valve was not diseased. The aortic valves were much thickened; they were covered over with loose vegetations, some being very large.

The aneurism was quite consolidated, being filled with laminated clot. It involved the femoral artery at a point just above and including the origin of the profunda. The fibres of the anterior crural nerve were spread out upon the sac.

At the seat of ligature there was a narrow band of white cicatricial tissue, in the centre of which at one point the unaltered silk ligature could be perceived. There was a little partially decolorised clot above and below the seat of constriction. Below this, and above the aneurism, there was about three-quarters of an inch of apparently unobliterated artery. (See the illustration at page 66.) The preparation is preserved in the College of Surgeons' Museum (No. in Catalogue 3241a).

Remarks.—It may be well to say that I use the term "spontaneous aneurism" to signify an aneurism which is not due to a penetrating wound of an artery. It would be difficult without some such arbitrary definition to say where spontaneous aneurism leaves off, and where the traumatic variety begins; for it is quite impossible to prove that external influences, such as sudden strains, blows, sudden or long-continued exertion leading to increased heart pulsation, are not largely concerned in the production of the so-called spontaneous aneurism, even in cases in which a morbid condition of the vessel is found.

The causes of such aneurisms and their exact mode of production are little understood. In my own case, the

cause was not by any means obvious. The aortic valves were much diseased, but no infarcts were found in any of the viscera after death. There was no history of rheumatism nor of syphilis, nor of any injury to the vessel either direct or indirect. Owing to the uselessness of the right lower limb, however, the left one was unduly worked, and might even occasionally be overstrained in consequence.

Whether, or how, this excessive use of the left limb may have contributed to the formation of the aneurism it is difficult to say. A marked feature in this case were the intensity and force of the heart's impulse; even after the boy had been kept at rest in bed for a week arterial pulsation was very violent and could be seen in all places, where the arteries were at all superficial. Such action was well calculated to put a severe strain on the arterial system, and if marked disease had existed at any spot it must almost necessarily have led to arterial dilatation. With regard to degeneration of the coats of arteries as a cause of this aneurism, a careful examination of the arterial trunks after death failed to detect disease at any spot (except the aneurism itself), nor did the microscope reveal any changes which the naked eye might have overlooked.

Concerning *age*, as a general cause of aneurism, the foregoing case is interesting on account of the youth of the patient. If we exclude the intracranial cases, spontaneous aneurism is rare in subjects under twenty years of age. In consulting the standard works on the subject I was at first led to imagine that even more cases had occurred than is actually the case. In the various tables of collected cases which have been published, the individual cases have doubtless been tabulated more than once, and hence the mistake.

With a view to greater accuracy I have tabulated all the cases of extracranial *spontaneous aneurism* occurring in subjects *under* twenty years of age, of which I could find *original records*, and as some of these records are not within easy reach, I have added to the table a very brief abstract of the notes of each of the cases.

Table of Fifteen Cases of spontaneous external Aneurism in persons under twenty years of age, compiled from the original records.

No.	Author, date, where recorded.	Artery: position.	Patient's Age.	Patient's Sex.	Condition of Heart.	Condition of Vessels.	Result.	Remarks.
1	Dr. James Sykes, 1823, Philadelphia Journ. Med. & Phys. Sciences, No. 11, May, 1823, p. 139	Carotid	18	F.	Not stated	Not stated	Cured by ligature	Fall from her horse two years previously. The tumour was of the size of a goose's egg. Made an excellent recovery after ligature of the common carotid. "A double animal ligature" was used, and the vessel tied in two places; the artery was not divided.
2	Sir A. Cooper, 1824, Lectures, edited by Tyrrell, vol. ii, p. 40	Ant. tibial	11	M.	,,	,,	Not stated	No details are given.
2A	Mr. Clive, 1824, quoted by Sir A. Cooper, op. cit., vol. ii, p. 63	Dorsalis pedis	Not stated	M.	,,	,,	,,	No details are given.
3	Moulinié, 1832, Journ. Hebdomad., vol. ix, p. 226	Ant. tibial (lower part)	19	F.	,,	,,	Died	Details are rather scanty. The aneurism became very large. Ligature of anterior tibial, which was undertaken on account of hæmorrhage, was followed by amputation. Death.
4	Dr. T. R. Smyth, 1834, Lancet, vol. i, p. 626	Ascending aorta	16	M.	Valves (aortic) diseased	—	Death	Walls of aorta were as thin as paper, soft and easily torn, but free from every kind of morbid deposit. The patient was seized suddenly with a sharp piercing pain in the region of the heart, after having ascended a ladder.
5	Mr. Grundy, 1834, Lond. Med. Gaz., vol. xiii, p. 509	Femoral (inguinal)	9	M.	Not stated	Not stated	Recovery	Followed on a kick in the thigh 18 months previously. Boy's health had been perfect up to the time of the onset of the tumour. The operation was difficult on account of the boy's struggles. The wound healed by the first intention.

No.	Author, date, where recorded.	Artery: position.	Patient's Age.	Patient's Sex.	Condition of Heart.	Condition of Vessels.	Result.	Remarks.
6	Dr. Miguel, 1835, Bull. Génér. de Thérapeut., vol. ix, p. 393	Aorta	14	M.	Normal	Not stated	Sudden death	The heart was of normal size; its orifices were free. The boy had been indisposed for 8 days only before the onset of acute symptoms, which were at first thought to be typhoid in nature. Before recording this case, Dr. Miguel remarks on the great rarity of aneurism in children.
7	Dr. Kingston, 1842, Edin. Med. and Surg. Journ., vol. lvii, p. 69	Int. carotid, near its origin. Basilar, close to its commencement	14 or 15	M.	Dilated; valves normal	Diseased	Death from embolism	Pulsation had been noticed for 2 years. A sudden onset of symptoms, including right hemiplegia and aphasia, which were considered as due to cantharides (but which were more probably due to embolism), led to his death in 2 days.
8	Prof. Syme, 1844, Lond. and Edinb. Monthly Journ. Med. Sci., Oct., p. 823	Popliteal	9	M.	Not stated	Not stated	Cured	Noticed 2 years before coming under Syme's observation. This author just refers to two other cases, but gives no details. He remarked on the rarity of the disease, and carefully discussed his diagnosis. He believed this to be the first case which had been "remedied by the modern operation at so early a period of life."
9	Dr. Armitage, 1857, Path. Soc. Trans., vol. ix (1857), p. 85	Aorta (bifurcation)	7½	F.	Healthy	Healthy	Died (suddenly)	No atheroma or roughness existed in the coats of the arteries. "The child had suffered from palpitations from her earliest infancy."
10	Dr. Ogle, 1865, Brit. and For. Med.-Chir. Rev., vol. xxxvi, p. 500	Ulnar	17	M.	Mitral valves diseased	Not stated	Death	Was admitted into St. George's Hospital with a pulsating swelling of the forearm. Sudden unconsciousness and aphasia supervened, and right hemiplegia. At the autopsy diseased heart and vessels were found, with extravasation of blood into substance of brain. Preparation is in the St. George's Museum (Catalogue, ser. vi, 129B).
11	Mr. Thomas Smith, 1867, Brit. Med.	Common femoral	12	M.	Aortic valves	Vessels healthy	Cured	The patient was a native of Wales; he was running after some horses in a field, when he experienced a

	Journ., March 16 (1867), p. 280				diseased			sudden pain in the groin. The ligature came away on 7th day; the artery where tied appeared quite healthy. Mr. Smith informs me that this lad died some years later of internal (? aortic) aneurism.
12	Dr. Norman Moore, 1882, Path. Soc. Trans., vol. xxxiv, 1883, p. 71	Ext. iliac (orifice of)	7	F.	Aortic and mitral valves diseased	Aorta itself normal	Not discovered during life	The preparation is in St. Bartholomew's Museum (No. 1460[a]), and is a true sacculated aneurism. The child died suddenly in the out-patient department. She had previously suffered from acute rheumatism.
13	Author's case, 1882	Common femoral	12 yrs. and 7 mos.	M.	Aortic valves diseased	Healthy	Death	Aneurism cured. The patient died after repeated and copious epistaxis. The preparations are in the Museum of College of Surgeons (No. in Catalogue, 3241A).
					Unpublished Cases.[1]			
14	Dr. [Sir Wm.] Gull, 1857, *vide* Guy's Hosp. Reports of Inspections, 1857, No. 127	Axillary	14	F.	Aortic valves "lacerated, retroverted, and covered with granulations"	Basilar artery and circle of Willis "were found healthy and pervious"	Death	Guy's Hospital Museum — Heart and Circulatory System, 1520[20]. The girl had never had rheumatism. Illness began 6 weeks before her admission to hospital. Left hemiplegia set in suddenly during sleep one night. She died some weeks later of a second attack of cerebral hæmorrhage. The aneurism was of the sacculated variety.
15	Dr. Habershon, 1872, *vide* Guy's Hosp. Reports of Inspections, 1872, No. 146	Femoral	16	M.	Mitral and aortic valves diseased	Diseased	Death	Guy's Hospital Museum — Heart and Circulatory System, 1519[21]. The lad had had rheumatic fever 12 months before his fatal illness. He died comatose after convulsions. At the autopsy, ulcerative endocarditis was found; heart weighed 15 ounces; cerebral arteries were much diseased; with secondary changes in the brain matter.

[1] I am indebted to Dr. Goodhart, Curator of Guy's Hospital Museum, for reference to these specimens, and for permission to make use of them.

Among alleged causes, and more especially as a cause of aneurism in young subjects, must be mentioned *embolism*. In analysing the foregoing table of cases, we cannot fail to notice the frequency with which valvular disease of the heart has been found coexistent with aneurism. Thus of the 15 tabulated cases in 2 cases only (Nos. 6 and 9) is the heart stated to have been healthy. In 5 cases (Nos. 1, 2, 3, 5, and 8) its condition is not recorded, while in 8, the remaining cases (Nos. 4, 7, 10, 11, 12, 13, 14, and 15) it is stated to have been diseased. These fifteen cases of extracranial aneurism are too few in number to allow us to arrive at any definite conclusion as to their cause or exact mode of formation. Even if we fall back on the much more numerous cases of intracranial aneurism (which, whether occurring in young subjects or in adults, are now generally believed to be due to embolism) the question cannot be positively settled. Nevertheless, the frequency of their association largely strengthens the view that embolism stands in some etiological relation to aneurism.

Since Dr. Kirkes first (in this country)[1] drew attention to this subject, many other papers have been published tending to confirm his views and to enlarge and point their bearing on aneurismal pathology.

Sir Wm. Gull,[2] in a paper "On Aneurisms of the Cerebral Vessels," says, "Whenever young persons die with symptoms of ingravescent apoplexy . . . the presence of an aneurism is probable. . ."

Mr. T. Holmes[3] relates a case in which the left internal carotid artery was affected by aneurism at the anterior part of the cavernous sinus. The tumour was as large as a small nut. The dilatation seemed to involve the whole circumference of the vessel, which was perfectly healthy in the rest of its course. The patient, a boy, æt. 16, was originally admitted into St. George's Hospital on August 21st, 1860, with heart disease, but unconnected with any

[1] In the 35th volume of our own Transactions.

[2] 'Guy's Hospital Reports,' 3rd series, vol. v, 1859, p. 281.

[3] 'Pathological Soc. Trans.,' vol. xii, p. 61, 1861.

history of rheumatism. There were clear indications of mitral disease and hypertrophy of the heart. He died some weeks later, and on examination a copious deposit of bead-like lymph was found lining the internal surface of the left auricle; and masses of similar lymph appeared also on the edges of the mitral valve. From the occurrence of embolism in the viscera, and the presence of vegetations on the valves, Mr. Holmes concluded that "embolism was probably the cause of the aneurism."

Dr. Ogle also contributed a valuable paper on this subject and case,[1] in which he argued to the same conclusion. More recently Dr. Church[2] published a little monograph "On the Formation of Intracranial Aneurisms in Early Life," in which he tabulates thirteen cases (including four under his own care) in subjects under twenty years of age; the arteries, without exception, are stated to have been free from atheroma or other disease; in seven of the cases the heart was diseased, and in six of these there were vegetations on the valves; in four of the cases the state of the heart is not mentioned. From these facts he concludes "that disease of the arterial wall is very rarely if ever a cause of cerebral aneurisms in the young." On the other hand "the extreme rarity of the disease renders it impossible that the co-existence of vegetations on the endocardium and aneurism of cerebral arteries can in all these instances be due to fortuitous circumstances." Dr. Church further emphasised these facts by contrasting them with cases of aneurism in adults. He examined fifteen consecutive cases in the dead-house of the hospital, and found the cardiac valves in every case quite free from vegetations.

Interesting cases of aneurism in young subjects, associated with valvular disease of the heart and infarcts in internal organs are also recorded in vol. xxviii of the Pathological Society's Transactions (1877) by Mr. Bryant and Dr. Goodhart. After discussing the subject the con-

[1] 'Medical Times and Gazette,' 1866, vol. i, p. 196.

[2] 'St. Bartholomew's Hospital Reports,' vol. vi, 1870, p. 99.

clusion was that "embolism and aneurism had the relation to each other of cause and effect" (loc. cit. p. 112).

The foregoing cases make it all but certain that embolism leads to aneurism, yet it is by no means easy to explain the exact mode in which the aneurism is brought about. Unfortunately there are no cases at our disposal, in which an aneurism is actually in process of formation, although there are many cases illustrating other effects of embolism—such as, dilatation of arteries above the seat of obstruction, and infarcts and their consequences in internal organs in all stages. Thus Mr. Shaw,[1] in describing a case of plugging of the right middle cerebral artery, says the artery was obstructed "by a firm white substance causing it to be stretched to about one third above its usual size." Mr. Joliffe Tuffnell[2] relates a remarkable case of dilatation of the popliteal artery. This dilatation during life had been regarded as "aneurismal," but it disappeared in large part after collateral circulation had been established. When the patient died the artery was examined. "No disease whatever could be found in the upper portion, but towards its lower end it had been converted into a fibrous cord of greyish-yellow colour, as solid as the walls of a long standing gristly stricture of the urinary canal."

These cases do not demonstrate the exact mode in which the aneurism is produced. For we cannot consider *dilatation of an artery* immediately *above* its obliteration, as in the cases just alluded to, *to be the same process as that which leads to the development of an aneurism in a vessel through which the circulation continues to go on.* Indeed, it is not a little remarkable, I think, that dilatation is not more common than appears to be the case, judging from the recorded instances that are to be found in our literature.

In Mr. Tuffnell's case the patient was very ill; he was suffering from ulcerative endocarditis, following a second

[1] 'Path. Soc. Transactions,' vol. vi, p. 34.

[2] 'Dublin Quarterly Journal,' May 1st, 1853, p. 371.

attack of rheumatism; he had likewise just previously contracted syphilis, for which he had been mercurialised. It is, therefore, by no means certain that arterial disease did not exist, although Mr. Tuffnell argued against it. It is specially worthy of observation that the "aneurismal development" occurred simultaneously with the embolism and that it disappeared within a few hours of the establishment of collateral circulation. Is it not possible that the embolism was arrested at the narrowest point of an artery, which was being gradually occluded by an arteritis and that the remainder of this softened area was dilated by the force of the blood-current and constituted the pulsating swelling observed during life? In one of Dr. Goodhart's cases (loc. cit. p. 108) it is stated that "the left brachial artery was considerably distended at its bifurcation, and on opening it a white creamy fluid escaped like pus, and having the microscopical features of pus. The lumen of the vessel was dilated and its walls soft. No clot was present in the vessel."

Dr. Goodhart argues that the "dilatation occurs not behind the obstruction, but in the obstructed spot." The mode of production he thinks is as follows: "The artery is plugged at a fork, and the clot gradually increases behind the original infarct, and as it increases it becomes squeezed together by the blood-pressure behind, and the artery is thereby dilated. Once weakened by dilatation, the remainder is easy to explain, we should rather expect an aneurism."

Dr. Goodhart, however, doubts whether this is the usual or only cause of dilatation, "because if so, all embolisms should show some such tendency to produce aneurisms, and they do not do so." He believes that the endocarditis is not the simple, but the ulcerating form of the disease, "generally producing fever and septic conditions. The clot detached from such a focus will poison the part in which it is lodged and lead to acute softening of the arterial wall."

If aneurism only occurred in association with ulcerative

endocarditis, such a view would deserve great attention, but this is not the case, as a glance at the records of the cases sufficiently shows.

Dr. Church (loc. cit. p. 106) says: "I think it probable that aneurismal dilatation takes place when the blood stream through the artery is obstructed, but not completely stopped." It would be less difficult to accept this theory, if aneurisms were only found in the smaller arteries; as, however, aneurisms occur in such arteries as the external iliac and in the femoral, I am inclined to regard this explanation also as insufficient. It is very difficult to explain how an embolon, sufficiently large to obstruct the femoral artery, could be lodged in the artery without giving rise to symptoms of arterial obstruction; whereas if the embolon be of small size, by what means is it arrested in its course, by what process does it become adherent to the vessel wall? It is urged that the plug is arrested at a bifurcation; but this is not by any means always the case.

In only two of the cases in the table is the aneurism stated to be of the sacculated variety (Nos. 12 and 14). Doubtless in such a case the aneurism may have commenced as a softening of that part of the arterial wall to which the embolon was adherent; but, in my own case at least, the artery is uniformly dilated, suggesting a uniform condition of the arterial wall, acted upon by some condition which was uniformly distributed over the whole calibre of the vessel.

In a paper entitled "Ueber embolische Aneurysmen, nebst Bemerkungen über das acute Herzaneurysma (Herzgeschwür)," Dr. Ponfick[1] recorded several interesting cases, and discussed the embolic causation of aneurism at some length. He found in some of his cases that the vegetations had become calcified, and that such calcified vegetations had been detached, washed into the circulation, and given rise to occlusion in some cases, to aneurisms in others; he argued that the sharp extremity

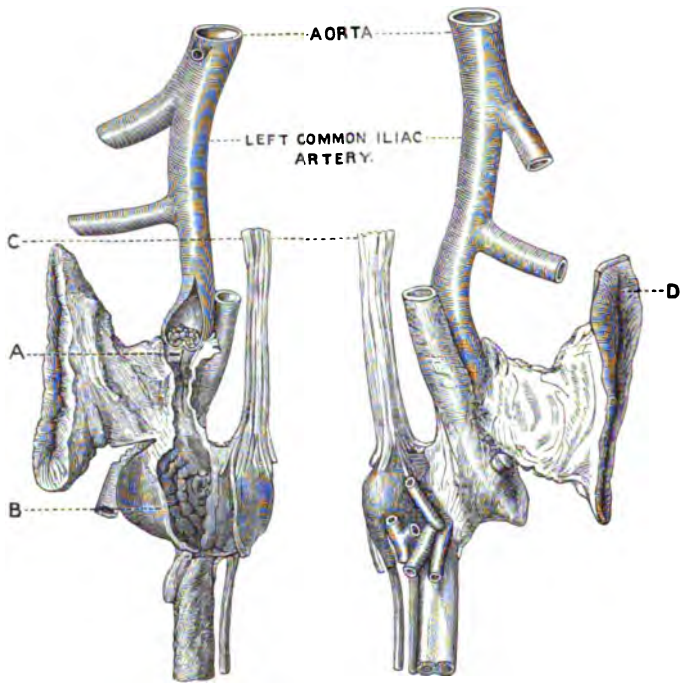
[1] 'Virchow's Archiv,' vol. 58, 1873, p. 583.

of a calcified vegetation had penetrated the intima, and, urged on by the constant thud of the circulating blood, had finally pierced the arterial wall. He gives drawings of two carefully dissected specimens, showing, as he thinks, this condition. The aneurisms are of the sacculated variety, and thus far support his contention.

Whether such a view as this is applicable to the majority of cases, I am unable to say. In my own case, it would not be tenable, for the vegetations were not calcareous.

For the present, therefore, while admitting that aneurism is frequently associated with embolism in the same subject, I do not think there is sufficient evidence to show that embolism is actually a cause of aneurism. Moreover, in the table of cases, there are at least two in which the heart is described as being *healthy*. It would seem probable (and be in keeping with many other pathological phenomena), that there are many factors at work. Apart, of course, from calcareous embola such as those described by Dr. Ponfick, I venture to think that *the condition which leads to plastic inflammation in one part of the arterial system, may produce local changes in other parts, which permit of the development of aneurism without the immediate intervention of an embolon.*

Aneurysm of Femoral Artery; Ligature of External Iliac; Death from Epistaxis.



A. Seat of ligature, a little partially decolorised clot above and below.
B. Aneurysmal sac, filled with laminated, partially decolorised clot.
C. Anterior crural nerve, spread over the sac.
D. External incision, through which the artery was approached.

INVESTIGATIONS

INTO THE

ACTION OF THE DIGITALIS GROUP.

BY

SYDNEY RINGER, M.D., F.R.C.P.,
PROFESSOR OF MEDICINE AT UNIVERSITY COLLEGE, LONDON,
AND
HARRINGTON SAINSBURY, M.D., M.R.C.P.,
PHYSICIAN TO THE NORTH-WEST LONDON HOSPITAL.

(Received October 4th, 1883.—Read November 21st, 1883.)

THE influence of digitalis on the circulation is, of course, the principal feature in the action of this drug. The primary slowing and increased vigour of contraction of the ventricle, the subsequent stage of irregular action as the dose is increased, and the final arrest of the ventricle in full systole are all well-known facts. To these must be added, rise in blood-pressure, an effect occurring uniformly, and which some authors consider of chief importance; certain, at least, of the cardiac effects of digitalis being, according to these, secondary to the increased blood-pressure. In a recent paper on the subject,[1]

[1] "Beiträge zur Kenntniss der pharmakologischen Gruppe des Digitalins," 'Archiv f. experimentelle Pathologie u. Pharmakologie,' Bd. xvi, p. 149.

Schmiedeberg thus classes the blood-pressure effects in relation to the cardiac effects:

1. Rise in blood-pressure accompanied, as a rule, but not necessarily, by slowed pulse rate.

2. Continuance of increased blood-pressure, with increased pulse frequence.

3. Continuance of raised blood-pressure with great irregularity in the heart's action.

4. Rapid fall in blood-pressure, stoppage of the heart; death.

Schmiedeberg attributes the therapeutic value of digitalis alone to this rise in blood-pressure; the slowing of the pulse, which is usually witnessed, he holds to be secondary to the rise in blood-pressure, a reflex on the heart through the vagi; the rise in blood-pressure itself, however, he makes dependent on the heart's action, and he further states that it is not attended by diminution in the calibre of the vessels. To a consideration of the relation between the heart's action and blood-pressure rise we shall return later on. What now concerns us rather is the condition of the heart and that of the arterioles. Concerning the former there is no difference of opinion, the ventricle is admittedly arrested in systole; concerning the latter the balance of evidence certainly appears to show that the arterioles contract under the influence of digitalis,[1] though Schmiedeberg, as above stated, denies this. But, allowing even that digitalis does cause the arterioles to contract, the question still remains, how does it do so? On the heart the action is generally admitted to be direct, but on the vessels it is held by most to be indirect, by the stimulation, viz. of the vaso-motor centres at the base of the brain. Ackermann almost alone maintaining that the action on the arterioles is direct (Wood, op. cit. p. 139).

The question is clearly one of considerable importance, and it bears directly on the definition of the digitalis action. Schmiedeberg, in the before-mentioned paper, enumerates a long list of bodies resembling digitalis in

[1] 'Consult Wood, 'Treatise on Therapeutics,' 3rd edit., pp. 138-139.

their action, and which he classes as digitalis remedies. The definition of such action being the primarily increased vigour of the ventricular contraction leading up finally to the systolic arrest, together with the blood-pressure rise. The question now, rather, is what is essential in this action, and whether we may not take as such the spasmodic condition of the heart, and accordingly define digitalis action as—*the production of continuous spasm of the heart muscle by direct action of the drug on this tissue:* this spasm not being of the nature of tetanus, *i. e.* of fusion of adjacent beats, and following on the application of the drug, either to the whole heart or to any limited portion of the ventricle, in the latter case causing a local spasm. Thus defined, Schmiedeberg's already very large group will have to be extended and made to include, among others, the caustic alkalies and barium chloride, acetate and nitrate,[1] and very probably many others.

Taking this as the definition of digitalis action, is it not probable that a drug thus directly acting on the muscular tissue of the heart will likewise affect directly the muscular tissue of the arterioles? Indeed, is one not justified in assuming that the likelihood of similar action of one drug on two or more tissues will be in proportion to the resemblance existing between these, approaching certainty as the tissues increasingly resemble each other, becoming less probable as they diverge? If this be allowed, the inference is not far from the heart to the arterioles, and given a particular action on the one, we should look for similar action on the other, and hence we should expect to find the digitalis (spasmodic) heart accompanied by spasm of the arterioles. This is, of course, on the assumption that the muscular elements of the arterioles may be influenced directly by the fluids bathing them. For the argument in evidence of this we must refer to Gaskell's paper on the tonicity of the heart and blood-vessels.[2]

[1] Boehm, "Ueber die Wirkungen der Barytsalze auf den Thierkörper," 'Archiv f. Exp. Pathologie und Pharmak,' 1875, iii, pp. 216, *et seq.* Also paper by the writers, 'Brit. Med. Journ.,' Aug. 11, 1883.

[2] Journ. of Physiology,' vol. iii, p. 48.

Whilst bearing in mind the resemblances existing between the muscular elements of the heart and arterioles, it is necessary that we do not lose sight of their differences. These differences are not merely structural, for functionally, whilst the muscular fibres of the heart and arteries resemble each other broadly, in that they are both forms of tissue specialised for the function of contracting, they differ widely in the mode in which they manifest this property. Contractility in the fully-differentiated organism, we find manifested after two modes, viz. after a slow persistent fashion and after a sudden explosive fashion. Non-striated and striated muscular tissues represent structurally the two extremes of this functional difference. The slow persistent contraction above named is closely allied, if not identical, with that condition we name *tonus*, which is specially characteristic of unstriped muscle. In striped muscular tissue, though the sudden contraction is the most striking characteristic, yet the slow persistent form is also present, and we speak of *tonus* for these muscles also. The heart muscle, according to physiologists, is intermediate functionally between the striped and the unstriped tissue; its contractions are more sudden than those of the latter, less sudden than those of the former. At the same time *tonus* is a marked feature of cardiac muscle. Concerning the element rhythm, which is characteristic of plain muscular fibre, and also to a marked degree of the heart, we do not find this intermediate position maintained.

Arguing, then, from this intermediate position of the heart with its affinities manifested in the two directions of the voluntary and involuntary muscles, we would suggest that it may be possible, from action on the heart, to infer in both of these directions; *e. g.* supposing that we find that the stress of action of a drug, applied to the excised heart, show itself mainly as affecting the character of the beat itself, we should then be led to infer that the drug will strongly affect voluntary muscular fibre, whilst, on the other hand, should it powerfully affect the tone of the heart, either in the way of increase or diminution, we should

then conclude that it will similarly affect the arterioles. It is essential to remember here that we are dealing with the excised heart, and hence are inferring from the local action on this tissue to the local action on other tissues.

We have made the above suggestion as to the possibility of inference from the heart to the voluntary muscles; the object, however, which we had principally in view was to establish experimentally the inference from the heart to the arterioles; and, accordingly, taking for our starting point the systolic heart of the digitalis group, we proceeded to examine the direct action of the members of this group on the vessels themselves.

Mode of experimentation.—This was the same as that adopted by Gaskell in his experiments on the direct action of acids and alkalies on the tonicity of the vessels (op. cit.), but here, as in our previous experiments with barium chloride, the tortoise was used instead of the frog. The exact method was as follows:—The animal was pithed, care being taken to destroy as completely as possible the spinal cord, the shell sawn through transversely and the soft parts divided. A cannula was then tied into the abdominal aorta distally, the hinder half of the body alone being used in the experiment. The cannula was fed with the required fluids by simple syphon action, and the flow accordingly was into the abdominal aorta and out through the cut abdominal veins. In order to measure the rate of flow, the preparation was placed on a tilted glass plate from which the escaping fluid drained into a graduated glass measure, by which means we were enabled to read off the amount of outflow for any given interval of time.

The pressure employed varied slightly in the different experiments, averaging about 30 centimetres, but, during each experiment it was maintained constant within 0·5 centimetre by continuous additions to the supply vessel, which was of large capacity, so as to retard changes in level.

The experiments were performed in May, June, and July of 1883. Each experiment lasted some considerable

time, two to three hours or even more, and inasmuch as the circulation of artificial saline fluids continued over lengthened intervals of time tends to produce œdema of the tissues, which œdema may even occasion considerable obstruction to the circulation,[1] it became necessary to try the effect of the circulation of the unpoisoned saline, over periods which would cover the duration of an ordinary experiment. By this means it was possible to see if the amount of *ischæmia* witnessed after the administration of a drug could be accounted for on the grounds of œdema alone.

Another point remained for consideration, viz. the vitality of the tissues. In the case, for instance, of a negative result, *i. e.* the addition of the drug to the circulating fluid remaining without effect on the rate of flow, the question arose whether the tissues were still vital and the arterioles still capable of contracting; this question was the more important the longer the experiment had lasted. We must here forestall the evidence as to the action of digitalis, and state that this drug acts most unequivocally and most strongly in causing the arterioles to contract, and that the effect comes out so rapidly that it is quite impossible it could be other than a vital effect. Hence, in this drug we had a means of determining the vitality of the tissues, for if at the end of an experiment, and after continued additions of the drug, the flow still remained unobstructed—the addition of digitalis to the circulation would at once determine whether the arterioles were still able to contract and so obstruct the flow. In the case of negative results, accordingly, this plan was adopted.

Two experiments were performed with *simple saline*,[2]

[1] See experiments by Glax and Klemensiewicz, 'Sitzungsberichte d. Akademie zu Wien,' vol. lxxxiv, Abth. iii, p. 216.

[2] The saline employed was a solution of 0·6 per cent. of sodium chloride in *tap* water. It is necessary to make this statement, since the small quantities of salts contained in ordinary tap water appear to exert important effects and make the distinction between such saline and that made with distilled water

we will quote the one showing the greatest variation in the rate of flow.

June 18th.—Temperature of room 17° C.; head of pressure 31 centimetres.

The following numbers were observed: outflow per five minutes in cub. cents. 80, 85, 95, 82, 88, 83, 78, 75, 70, 60, 68, 66, 65, 65, 65, 77, 79, 78, 80, 80, 77, 80, 83, 80, 82, 86, 79, 84, 83, 86, 80, 80, 84, 81, 79, 83,[1] 35, 15.

[1] Digitaline solution, 1 per cent., 1 c.c. added to the 200 c.c. of saline.

The second experiment showed a considerably more uniform rate of flow than that here seen; but the important point to note in the above experiment is that at the end of three hours the rate of flow is undiminished, and that at this time the vitality of the tissues is still maintained, as is manifest by their immediate reaction to digitaline, the flow lessening at once from 83 c.c. to 35 c.c. in the next interval of five minutes, and thence to 15 c.c. Exactly similar results obtained for the second experiment, the rate of flow at the end of three hours was at the highest figure recorded during the experiment, and from this, viz. 48 c.c. it fell, on the addition of digitaline, at once to 25 c.c., and in the five minutes following this to 4 c.c.

Digitaline.—The digitaline employed was obtained from Morson. The above two experiments both show the effect of digitalis in obstructing the circulation and the rapidity with which this is effected. Of three other experiments, made simply with a view to test the action of digitaline, the results were in each case similar. We will quote only one.

May 24th.—Temperature of room 24° C.; head of pressure 31 centimetres.

Outflow per five minutes in c.c.—90,[2] 88, 80, 84,[3] 48,

[2] Saline solution, 0·6 per cent. supplied. [3] Digitaline, 1 per cent., 0·5 c.c. added to the 100 c.c. saline.

a very essential one, *vide* Ringer, "On the influence exerted by the Constituents of the Blood on the Contraction of the Ventricle."—'Journal of Physiology,' vols. iii and iv,

28, 12,[1] 6, 9, 17, 24, 31, 34,[2] 25, 10, 4,[3] 2, 2, 2, —, —, 11.

[1] Saline substituted. [2] Digitaline saline again supplied. [3] Saline again substituted.

The evidence of the other experiments was precisely to the same effect, and they are omitted simply on account of space. In the above experiment the alternation of effect is to be observed, *e.g.* with the supply of the digitalis saline the rate of flow at once begins to fall rapidly, on resubstituting pure saline it slowly begins to rise.

It is to be noted that the minimum is, in the above, reached after the replacement of the poisoned circulation by saline, this of course simply means that the digitaline had not had full effect and that the process of washing it out was not an immediate one. The blank spaces in the last section simply represent four intervals of five minutes allowed to elapse before again measuring the rate of flow, the last figure shows this to have been slowly increasing.

Barium chloride.—For the details of the experiments with this drug we must refer to our paper in the 'Brit. Med. Journ.' for August 11th, 1883. The method adopted in these experiments was precisely the same and the results equally unequivocal. *Barium chloride acts strongly and directly on the small arterioles, causing these to contract.* It may be remembered that Boehm[4] had previously demonstrated the digitalis-like action of the salts of this base in the production, viz., of the retarded pulse-rate, the heightened blood-pressure, and the systolic heart.

Strophanthus (the African arrow poison, Strophanthus kombi).—Two experiments were made with this drug. A 5 per cent. solution of the crude drug was employed. The results were much less striking than for digitaline, still they were in the same direction as for this latter. We will quote one experiment.

[4] "Ueber die Wirkungen der Barytsalze auf den Thierkörper," 'Archiv für experimentelle Pathologie u. Pharmakologie,' 1875, iii, pp. 216, 251.

May 28th.—Temperature of room 20° C.; pressure 31 centimetres.

Rate of outflow per five minutes in c.c.—42,[1] 39, 41, 41,[2] 37, 28, 25, 27,[3] 23, 24, 22, 18, 16, 10, 10, 11, 11, 12, 15, 16, 18, 20,[4] 20, 11, 9, 10, 9, 10, 10, 8, 9, 7.

[1] Saline solution, 0·6 per cent. supplied. [2] Strophanthus 5 per cent., 0·4 c.c. to 100 c.c. saline. [3] Saline solution replaced. [4] Strophanthus 5 per cent., 1 c.c. to 100 c.c. saline.

It is to be noted that in this experiment, though the effect of the strophanthus is much less sudden and much less marked, yet that, following the addition of the strophanthus, there is a steady unbroken fall, continuing it is true long after the replacement by saline, but again reappearing after the rate of flow had begun to mount, on the readdition of strophanthus.

The second experiment gave a very sudden fall of the rate of flow following the first addition of strophanthus; throughout the remainder of the experiment the rate of flow, during the periods of saline substitution, never rose to within one half the initial rate of flow, and on this lowered rate the subsequent additions of strophanthus gave no decided effect in further reducing it.

Scillitine.[5]—Two experiments with a one per-cent. solution were made but the results were not decided, and they scarcely permit of more than the negative conclusion that the action if of the same kind as that of digitalis, comes very far indeed behind it in degree. Large doses were

[5] We are indebted to Mr. A. W. Gerrard, F.C.S., Lecturer on Pharmacy at University College Hospital, for the following account of his method of preparation of the scillitine employed in our experiments: "Five pounds of squill bulb were exhausted with proof-spirit, and the resulting tincture treated with four times its volume of 84 per cent. alcohol; this caused the precipitation of a viscous, tenacious extract, the spirit retaining in solution the scillitine. The alcohol was removed by distillation and the residue treated a second time with alcohol and the alcohol again distilled off. A pale green extract was thus obtained which was washed with ether to separate the green substance, and further diluted with water to precipitate resin and colouring matter. The clear aqueous solution was now evaporated and gave a pale scaly mass of scillitine, answering to the reactions of a glucoside."

employed, viz. as much as 5 c.c. of the one per-cent. solution to the 100 c.c., and the contrast between the comparatively slight action of this substance and the very striking and sudden effect produced by the digitalis in one fifth the dose, was very marked. The digitalis was tried in the end stages of both of the scillitine experiments, and the rate of flow fell almost at once to a minimum, which practically amounted to complete obstruction.

Dyak poison.—This is an arrow poison used by the inhabitants of Borneo, concerning the origin of which nothing certain is known, but it contains in all probability the juice of *Antiaris toxicaria* as a chief constituent.[1] It is a heart poison and acts in this respect like digitalis. Two experiments were made with this substance, we will quote one of these which, in addition to illustrating the influence of the dyak poison, shows well the relative action of digitaline. The solution strength was a one per-cent. of the crude drug.

June 15th.—Temperature of room 19° C.; head of pressure 31 centimetres.

Rate of outflow per five minutes in c.c.—72,[2] 74, 76, 71,[3] 70, 63, 55, 48, 39, 37, 37, 36, 32, 36, 36,[4] 36, 42, 40, 44, 46, 46, 46, 48,[5] 47, 47, 47, 46, 45, 45, 47, 48, 44,[6] 43, 43, 40, 40, 36, 36,[7] 24, 4.

[2] Saline solution, 0·6 per cent. supplied. [3] Dyak poison 1 per cent.; 0·5 c.c. to 100 c.c. saline. [4] Saline solution resubstituted. [5] Dyak poison 1 per cent.; 1 c.c. to 100 c.c. saline. [6] Dyak poison 1 per cent.; 4 c.c. to 100 c.c. saline. [7] Digitaline 1 per cent.; 1 c.c. to 100 c.c. saline.

This experiment shows in the first instance a decided fall on the administration of the poison, from this fall the recovery with the saline is but slight, but the further addition of the poison remains practically without effect, though in the last instance the dosage is increased considerably. The last two figures show well the powerful relative effect of digitaline and they further demonstrate the persistence of vitality in the tissues after over three

[1] See Husemann, 'Vergiftungen,' p. 482 (Maschka's 'Handbuch d. Gerichtlichen Medicin,' vol. ii).

hours of artificial circulation. The second experiment gave results more decided in favour of the dyak poison, both the first and the second additions of the poison reducing the rate of flow by one half; still it places this poison far behind digitaline in its direct action on the vessels.

Convallamarin.—This is a glucoside obtained from *Convallaria majalis* and is classed among digitalis remedies[1]. But one experiment was made with this substance; we quote it as follows:

June 21st.—Temperature 17·5°; head of pressure 31 centimetres.

Rate of outflow per five minutes in c.c.—22,[2] 22, 24, 29, 33,[3] 23, 22, 24, 19, 16, 16, 16, 18,[4] 18, 22, 25, 26, 29, 30, 33,[5] 29, 24, 20, 17, 16, 15, 17,[6] 15, 17, 17, 16, 16, 16,[7] 16, 3, 1.

[2] Saline solution, 0·6 per cent. supplied. [3] Convallamarin 1 per cent.; 0·4 c.c. to 100 c.c. saline. [4] Saline solution replaced. [5] Convallamarin 1 per cent.; 0·8 c.c. to 100 cc. saline. [6] Saline solution replaced. [7] Digitaline 1 per cent.; 1 c.c. to 100 c.c. saline.

The results are not very striking, but it will be observed that the first addition of convallamarin reduced the rate by almost one half, viz. from 33 c.c. to 18 c.c.; that saline replacement brings back the previous rate of flow 33, and that convallamarin again reduces this rate by one half; the subsequent addition of saline does not show recovery. The last three figures show the immediate and marked effect of digitaline.

It will be remembered that Gaskell (op. cit.) demonstrated the effect of alkalies on the arterioles of the frog,

[1] Mr. Gerrard kindly prepared this substance for us and as follows: "The plant was exhausted with rectified spirit and the spirit distilled off; the residual extract was dissolved in water and evaporated to a thick fluid, then treated with ten times its volume of alcohol, filtered, and evaporated. The residue was now treated with tannin in excess, to form tannate of convallamarin, which after purifying by washing with alcohol was rubbed up with a little water and lead oxide—tannate of lead and free convallamarin being the result; the latter was removed by alcohol and obtained as a straw-coloured scale on evaporation."

this effect being constriction even to the extent of complete obstruction in some cases. The reverse obtained for acids, *i.e.* the vessels dilating under the influence of the acid. Dr. Gaskell employed solutions of sodium hydrate and of lactic acid to demonstrate these effects. We have repeated these experiments on the tortoise for the following alkaline salts; potassium hydrate, carbonate and bicarbonate, sodium carbonate and bicarbonate. The potassium salts gave us like results as also did the sodium carbonate; in all cases the vessels became constricted. With the sodium bicarbonate, however, we did not get this result, the flow instead of diminishing in rate appeared, if anything, to actually increase. This result, moreover, was obtained although the sodium bicarbonate was employed in such doses as to produce a deeper blue with test-paper than did the sodium carbonate. What this may mean exactly it is difficult to say, but if the ability to turn red litmus paper blue be accepted as the definition of alkalinity then an exception must be made in favour of sodium bicarbonate in respect of the statement that alkaline salts increase vascular tone. The power of a salt to saturate an acid obviously cannot be taken as a measure of its alkalinity since a molecule of sodium hydrate and of sodium bicarbonate would for all the stronger acids show an equal capacity in this respect, yet the latter is a comparatively saturated salt as contrasted with the former. We have laid the greater stress on this point because Gaskell has drawn attention to the acid and alkaline effects as rather fundamental differences and has suggested that all bodies may show a general division into acid-, and alkali-like actions; thus he suggests that antiarin and digitaline would come under the latter heading, muscarin under the former. However, with this exception of sodium bicarbonate the results obtained on the tortoise with the remaining alkaline salts confirm those obtained by Gaskell on the frog.

Simultaneously with these experiments on the vessels others were made on the excised frog-heart. In these an

artificial saline solution was circulated through the heart tied on to the perfusion cannula of a Roy apparatus by a ligature placed in the auriculo-ventricular groove. To this solution was then added the particular drug under experiment. The results were as follows:—Digitaline, strophanthus, dyak, scillitine, convallamarin, all caused persistent spasm of the ventricle, in varying degree however, digitaline being by far the most active in this respect. The experiments on barium chloride have already been given elsewhere ('Brit. Med. Journ.' Aug. 11th, 1883); they too showed persistent spasm as the result of direct action of the drug on the heart. Similarly experiments made in 1882 with the hydrates and carbonates of potassium and sodium,[1] showed persistent spasm to result from direct action on the excised ventricle, with the notable exception, however, of sodium bicarbonate which is there recorded as not producing persistent spasm. This exception then would accord with the results obtained for the vessels.

In the last-mentioned experiments it was noted that the bicarbonate of potassium caused much less persistent spasm of the ventricle than did either the carbonate or hydrate. These experiments were repeated now with like results. It was, however, observed in addition that the adding of sodium bicarbonate to a saline solution not containing a potassium salt (in physiological dose) did cause persistent spasm. This at first led us astray, showing as it appeared to do a difference in the action of the sodium bicarbonate on heart and vessels. When, however, we made the experiments exactly comparable by adding a physiological quantity of a potassium salt to the pure saline, or instead of this employed saline made with tap water in place of distilled water (the former containing the necessary trace of potassium salt), when this was done the discrepancy disappeared, and the heart behaved just as the vessels had done. It must be remembered that the saline fluid employed to circulate through the vessels was made with tap water.

[1] *Vide* 'Practitioner,' Aug., 1883, "On the individuality of action of the component parts of a drug."

We did not mention aconitia among the foregoing experiments on the tortoise; this drug, however, was tried with the result of apparently causing dilatation of the vessels. When, however, tested on the excised frog's heart an opposite result obtained, viz., spasm of the heart muscle. We suggest that the following may possibly explain this seeming contradiction; the heart possesses the property of rhythmic contractility, and we have elsewhere shown that, where there is a tendency to persistent spasm of the ventricle, the rhythmic contractions accentuate their spasm or appear to develop it. In the present experiments on the heart with aconitia the spasm was in connection with rhythmic contractions, and when these were prevented as by ligaturing the ventricle through its upper third, the spasm did not develop. Should this explanation be the true one, this second discrepancy would be apparent only.

Thus far we have seen the direct action on the vessels to be similar to that on the heart; there yet remains for determination the question as to whether any of the effect on the vessels is to be attributed to nervous agency, *i.e.*, whether in addition to direct action on the vessels, these may be influenced reflexly through the nervous system. The following experiment was designed to answer this:— The cerebral and optic lobes were destroyed, care being taken to ensure there being the entire thickness of the cerebellum between the surface of section and the fourth ventricle. We may thus take it that the vaso-motor centre was uninjured. From the brainless tortoise the whole abdominal shell wall was then removed, and the peritoneum carefully separated up from the shell wall; it was then opened longitudinally, the descending aorta exposed, ligatured proximally and a cannula tied in distally. The large vein running up in front of the lung to the liver was then exposed and ligatured proximally; the same was done for the two large veins in the anterior abdominal wall. All three veins were then opened distally, and the saline solution started as for the previous experiments.

By the means adopted a free outlet from the veins was provided for the inflowing saline, and the escaping fluid could be caught and measured as in the foregoing experiments. In addition to this the vessels through which the saline was flowing were under central nervous control, since the spinal cord, including the medulla with its vaso-motor centre, was intact. The poison was then injected into the muscles of the back of the neck.

Two experiments were made with digitaline; of these the first gave purely negative results, just as occurred in two out of the three experiments made after the same method with barium chloride. The digitaline when injected into the muscles remained without effect on the saline circulation, and even when added to the saline solution itself it failed to produce any very definite effect. The second experiment gave similarly a negative result for the injection of the poison into the anterior circulation, but on adding the drug to the saline circulation the tissues at once reacted. We record the details of this experiment.

June 27th.—Temperature 18°; head of pressure 30 centimetres, maintained constant within 0·5 centimetre.

Rate of outflow per five minutes in c.c.—29,[1] 29, 26, 29, 30, 32,[2] 33, 33, 37, 42, 43, 48,[3] 46, 52, 50, 46, 55, 53,[4] 43, 12, 2.

[1] Simple saline solution (made with tap water), 0·6 per cent. [2] Digitaline, 1 per cent. 1 c.c. injected into muscles of neck in 0·5 c.c. doses. The ventricular contractions seemed to increase in vigour, but the diastoles remained good, or even more complete. Rhythm somewhat irregular. [3] Digitaline, 1 c.c. injected in 0·5 c.c. doses, as above. Both diastole and systole remained good. [4] Digitaline, 1 c.c. *added to the saline circulation.*

The brain cavity after completion of the experiment was more completely opened and the brain found to have been destroyed at the level of the posterior part of the optic lobes; cerebellum apparently intact.

Assuming the poison to have been absorbed then it must have been pumped by the heart to the vaso-motor centre, yet it appears to have been unable to influence the calibre of the vessels of the hinder extremities, which

latter the final addition of digitaline to the saline circulation itself proves to have been ready to respond.

The very free vascular anastomosis which obtains in the tortoise may at first sight appear to invalidate the experiment, since it scarcely allows of complete isolation of the two circulations. This objection would certainly hold had we obtained a positive result, *i.e.* had the injection of the poison into the anterior circulation caused contraction of the vessels of the posterior circulation, since the poison might have entered the latter through the anastomoses; it can, however, scarcely stand for a negative result.

Finally, there remains for us to record some experiments made on warm-blooded animals with digitaline.[1] This drug alone was selected as typical of the group, and the warm-blooded animals were experimented on in order to complete the series and bring our results a step nearer to human physiology.

The apparatus we employed was very similar to that used by Glax and Klemensiewicz in their experiments on artificial circulation.[2] A large air-chamber in connection with a force-pump allowed us to get up pressure. The efferent tube from the air chamber was put in connection with two three-necked Woolf bottles by a T tube, both arms of the T tube being provided with clamps. Into each of the Woolf bottles a mercury manometer was inserted, and, finally, from the third neck of each bottle an efferent tube was brought into connection by means of a T tube with the cannula for the aorta. Each afferent tube was provided with a clamp. Each Woolf bottle was of large capacity and was filled with the saline fluid for circulation. Both stood in a large water-bath, which was maintained at a constant temperature of 39° to 40° C. The use of the two bottles was to allow of the filling of the spent bottle without stopping the flow or altering the pressure. And

[1] We are indebted to Professor Schäfer for having kindly performed these experiments for us.

[2] 'Beiträge zur Lehre von der Entzündung. Sitzungsberichte der Wiener Akademie,' Bd. lxxxiv, Abth. iii, p. 216.

this was readily managed by simply clamping off the tube leading from the pressure bottle to the spent Woolf bottle and opening the tube leading to the other, and similarly opening the efferent tube from the latter and closing that from the spent bottle. By keeping a large stock supply of the artificial circulating fluid also immersed in the bath we were able to replenish the bottles whilst maintaining the temperature of this fluid nearly constant. We will first of all give an experiment in which the fluid was not poisoned, the saline circulation being maintained right through. This gave us our control experiment and showed us the limits of variation independent of drug action.

July 13th.—Temperature of room 23° C.; experiment on a cat.

Temperature of bath 39°—40° C.; artificial circulating fluid = simple tap water saline 0·6 per cent. + a physiological trace of potassium chloride, viz. 0·7 c.c. of a one per-cent. solution to the 100 c.c. saline. Pressure 100 mm. Hg. The animal was chloroformed, the abdomen opened by an incision in the linea alba, the inferior cava and descending aorta exposed and both vessels ligatured proximally. Into the descending aorta the afferent cannula was then inserted distally and tied in; into the inferior cava the efferent cannula was likewise inserted distally and tied in. The experiment was then started.

During the whole experiment up to the death of the animal narcosis was maintained so that the experiment was painless.

Rate of outflow per five minutes in c.c.—300, 240, 310, 380,[1] 415, 380, 335, 320,[2] 265, 155, 170,[3] 170, 190, 180, 200,[4] 175, 175, 150,[5] 130, 130, 120,[6] 115, 100, 85,[7] 75, 65,[8] 100,[9] 120, 110.

[1] No œdema so far. [2] Animal dying. Doubtfully slight œdema of tissues. [3] Animal dead. Distinct œdema. [4] Œdema marked. [5] Leakage from the tissues exposed in the wound, especially from the surface of the intestines. The leakage is due to simple sweating through the tissues. [6] Skin of legs quite tense from the œdema. [7] Skin very tense. [8] Free incision through skin and subcutaneous tissue of one leg. [9] Free incision into other leg.

The experiment teaches several things; in the first place the rather considerable variations in the rate of flow in the early stages before the change was in any particular direction. Then with oncoming œdema the steady and somewhat rapid fall in the rate of flow till from a rate of flow of from 300 c.c. to 400 c.c. per five minutes it has fallen to 65 c.c. in the five minutes. That this effect was largely the result of the œdema, we learn not only from the advance of the two *pari passu*, but also from the effect of the free incisions, which lessening tension at once allowed a freer circulation.

The figures representing the rates of flow subsequent to the incisions are certainly lower than they should be, and probably decidedly lower since from the large surface of the incision considerable oozing took place which in part would represent escape through some of the smaller cut vessels, and by this quantity the above numbers would be deficient.

To contrast with this we give the following experiment.

July 9th.—Temperature of room 20° C.; experiment on rabbit.

The same apparatus was employed, but the stock supply of saline was not so large as in the previous experiment, and the through flow was so rapid that, though the bath was maintained at a constant temperature of about 40° C., the saline supply must have been below this temperature, therefore the temperature conditions were less satisfactory. The saline was of the same composition as in the previous experiment, the pressure in the manometer was 50 mm. Hg.

The animal was kept under chloroform throughout the experiment.

Rate of outflow per five minutes in c.c.—178, 188, 174, 270, 310,[1] 102, 80, 16, 14,[2] 8, 8, 9, 10, 11, 13,[3] 13, 14, 14, 16, 16, 16, 19, 20, 16, 19,[4] 17,[5] 17, 14.

[1] Digitaline 1 per cent., 1 c.c. to 100 c.c. saline. [2] Unpoisoned saline resubstituted. [3] Animal dead. [4] Marked œdema. [5] Incision into subcutaneous tissue to try and free the vessels.

Making all allowance in this experiment for the temperature not being quite constant, the results are yet too striking to admit of any other interpretation than that the digitaline did act directly and powerfully on the vessels. If the numbers be compared with those in the previous experiment this will be at once apparent. It will be observed that the resubstitution of saline did not appreciably improve the circulation; with reference to this, however, it must be remembered that the rate of flow at the time of resubstitution was exceedingly slow, and hence it would naturally take a long time to wash through the vessels; further, that in the later stages the increasing œdema was an obstacle to improvement, and finally, that the tissues of warm-blooded animals are much more delicate, and hence less likely to recover from damage done. That the incision into the tissues should be without effect will be comprehensible on similar grounds.

Another experiment on a cat was made in which, after establishing a saline flow, digitaline was injected into the subcutaneous tissue, of the anterior or heart circulation. The results obtained were negative, but the apparatus employed was rather less convenient than the above described, and various sources of fallacy crept in.

Confining our attention, however, to the two experiments just described, we find the results in keeping with those already obtained with cold-blooded animals, and we think the present series of experiments establishes tolerably definitely the direct action of digitalis and the group of bodies of which it is the type on the vessels.

To complete the argument which we stated at the commencement of the paper, experiments were made to ascertain the degree of poisonous action of these drugs on the skeletal muscles. Definite quantities of the drugs were injected beneath the skin of the back in frogs, and subsequently to systemic death, or in some cases even before, the muscles tested electrically as to their excitability.

The following were the results obtained:

Digitaline. 1 per cent. solution.

Weight of frog.	Quantity injected.	Condition of muscular irritability.
34 gms. ...	0·3 c.c.	... Systemic death in 3½ h.; muscular irritability gone in 6 h.
18·5 „ ...	0·2 c.c.	... Muscular irritability gone in 5 h. 30 m.
31·5 „ ...	0·2 c.c.	... Frog quite well after 19 h.
16·5 „ ...	0·2 c.c.	... Muscular irritability gone in 4 h. 45 m.
11 „ ...	0·2 c.c.	... Muscular irritability gone in 3 h. 25 m.
? „ ...	0·4 c.c.	... Systemic death in 1 h. 20 m.; muscular irritability fair after 3 h. 25 m.

Strophanthus. The crude drug.

25·5 gms. ...	0·2 c.c. (2·5 %)	... Lost in 2 h. 20 m.
25·75 „ ...	0·2 c.c. (1 %)	... Lost in 2 h. 40 m.
14·75 „ ...	0·2 c.c.	... Lost in 2 h. 30 m.
10·75 „ ...	0·2 c.c.	... Lost in 2 h. 40 m.
32 „ ...	0·2 c.c. (5 %)	... Lost in 1 h. 10 m.
12 „ ...	0·2 c.c.	... Nearly gone in 1 h. 30 m.
10·5 „ ...	0·2 c.c.	... Nearly gone in 1 h. 30 m.
? „ ...	0·4 c.c.	... Quite gone in 2 h. 45 m.

Dyak. The crude drug; but one experiment.

22 gms. ...	0·2 c.c. (1 %)	... Lost in 1 h. 20 m. (Systemic death in 50 m.)

Scillitine.

31 gms. ...	0·2 c.c. (1 %)	... No effect; frog normal.
38 „ ...	0·3 c.c. „	... No effect; frog normal.
45 „ ...	0·5 c.c. „	... Muscular irritability lost in 6 h. Systemic death in 2 h.
18·5 „ ...	0·2 c.c. „	... Muscular irritability nearly gone after 20 h.

Barium chloride.

23 gms. ...	0·5 c.c. (10 % solution)	... Lost in 3 h. 15 m.
19·75 „ ...	0·3 c.c. „	... Lost in 3 h. 15 m.
20·75 „ ...	0·2 c.c. „	... Lost in 3 h. 15 m.
20·75 „ ...	0·3 c.c. „	... Still slight persistence after 3 hours 15 minutes.

Weight of frog.	Quantity injected.	Condition of muscular irritability.
23·75 „	... 1 c.c. (1 % solution)	The frogs developed within the first few hours peculiar stiff movements and the cry-reflex described by Boehm; from this state they for the most part recovered, and at the end of 24 hours were fairly normal.
24·75 „	... 0·5 c.c. „	
27·5 „	... 0·5 c.c. „	
?	... 0·2 c.c. „	... No effect.

Examining these results we observe in the first place that the above drugs from the digitalis group are all *notable poisons to the skeletal muscles.* And we observe, further, that strophanthus and dyak poisons act most powerfully in this respect. It is true that some of the experiments with strophanthus were with 2·5 per cent. and 5 per cent. solutions as against 1 per cent. of digitaline, but then we must bear in mind that of the former we were employing the crude drug as against the active principle itself in the latter case. Hence it is clear that both of these drugs, strophanthus and dyak, affect more powerfully the voluntary muscles than does digitaline. If, now, we refer back to their action on the heart we shall find that whereas the chief feature in the digitaline action is the contracture excited, which may be almost complete with scarcely any weakening of the heart's action, in the case both of strophanthus and dyak we find that though contracture is well-marked it is less so than with digitaline, whilst at the same time there is more decided weakening of the individual beats. For scillitine the results are less decided; in its action on the skeletal muscles, it scarcely exceeded digitaline in power, whilst on the heart it caused less marked contracture, but decided weakening of the individual beats.

With these results in view we are able to speak less positively as to the possibility of inferring from the action of these cardiac drugs on the heart contractions to action on the skeletal muscles themselves. To determine this point a more extended range of experiments will be needful.

We may now briefly re-state the steps of the argument and sum up the results obtained.

1. Starting from the systolic digitalis heart, which is admittedly a result of direct action on the cardiac muscle, we have found:

2. That for the other members of the digitalis group here examined a like direct action obtains.

3. We have suggested that this local action on the heart, which may affect either the whole heart or a limited portion, according as the drug comes in contact with the whole heart or a limited portion of the heart, that this action may serve as the definition of *digitalis action*, and that such definition would necessarily enlarge Schmiedeberg's already large gronp.

4. Taking this as the definition we have argued therefrom to local action on the vessels, and have found experimentally such to hold—the digitalis group causing persistent spasm of the arterioles.

5. We have endeavoured to ascertain if the calibre of the vessels may be influenced by these drugs *indirectly* through the nervous system, and, so far as our experiments go, have been answered in the negative.

6. Finally, we have found by experiment that the drugs digitaline, strophanthus, dyak, scillitine, and barium chloride, are markedly poisonous to the skeletal muscles.

The results require some criticism. In the first place, we note that the definition adopted admits into the group such bodies as the caustic alkalies, barium salts, &c. The former certainly do not rank with us clinically as digitalis-like drugs. But it is obvious that clinical use is practical use. And that it must very extensively weed this large scientific group, for it is at once clear that it is practically impossible for us to give alkalies to the extent of alkalising the system to the degree requisite to bring out the digitalis-like action. Hence the alkalies would be excluded from the clinical group. It is very possible that for similar reasons the barium salt may prove impracticable, and that the gastro-intestinal irritation (vomiting

and purging) excited by the drug, may not allow of its being given in dose sufficient to bring out the cardiac and vascular effects. This point, however, will have to be determined by experience. The like may obtain for many other drugs yet to be included in the digitalis group as above defined, for clinical value is a question of fitness, and results from the fact that the body is an assemblage of organs, and that the action of a drug in many cases is simultaneously on several of these; hence whilst its action on one organ would qualify it for clinical use, by its action on another organ it disqualifies itself.

One other point we would wish to touch upon, viz. the question of the rise of pressure and its dependence on cardiac action. According to Schmiedeberg the blood-pressure rise is due solely to increased cardiac action. "It has been shown," says Schmiedeberg, "to be not attended by diminution in the calibre of the vessels."[1] In view of our own experiments and also of those by Donaldson and Stevens, the results of which have been recently published[2] (subsequently, however, to the completion of our own experiments both on barium chloride and on the digitalis group), we can hardly accept this statement and hold that part at any rate of the pressure rise must be attributed to narrowing of the calibre of the arterioles. This interpretation indeed appears to us much more in consonance with the data which Schmiedeberg himself furnishes us with, for in the above paper he gives rise of blood-pressure as continuing through the first stage in which there is as a rule slowed pulse-rate, through the second stage in which there is increased pulse frequence, and through the third stage in which there is great irregularity in the heart's action; in the fourth stage, however, with systolic arrest there is rapid fall in blood-pressure. Here we see increased blood-pressure obtaining with most varied heart action, and we can scarcely imagine that in the third stage the cardiac action can be effective in main-

[1] Op. cit.

[2] 'Journal of Physiology,' vol. iv, pp. 165, *et seq.*

taining the increased tension. Arguing from this we should rather be tempted to exclude the heart and refer all to the vessels; a conclusion, however, which we think would necessarily be faulty, for is not the question one of action and reaction—the heart acts on the arterioles which again react on the heart? It is quite clear that without a peripheral resistance you could never whip up blood-pressure just as clearly as that with no matter how great a peripheral resistance (provided there were still means of outflow, *i.e.* that the obstruction were not complete) raised blood-pressure could never be maintained with a failing heart. Donaldson and Stevens more especially deal with the question of the work of the heart, though they also take up the question of the condition of the blood-vessels; these latter results being similar to our own. As to the work of the heart they find it diminished. We would, however, suggest that it is possible this resulted from their having pushed the drug too far, for in their experiments on the terrapin, where they record lowered work and also the condition of the heart, we note of this latter that it is very markedly affected; thus, the ventricle is either distinctly shrunken, or there is marked irregularity in its action, or dicrotism, or many auricular beats to one ventricular. In this stage we should scarcely look for increased work of the heart, but should feel rather that we were bordering on stage 4 of Schmiedeberg with fall in blood-pressure.

ON

HIGH AMPUTATION FOR SENILE GANGRENE.

BY

JONATHAN HUTCHINSON, F.R.S.,

EMERITUS PROFESSOR OF SURGERY TO THE LONDON HOSPITAL, AND PRESIDENT OF THE OPHTHALMOLOGICAL SOCIETY.

Received October 9th—Read December 11th, 1883.

My wish in writing the present paper is to claim the attention of the Society to the advantages of amputation high up in all suitable cases of senile gangrene. The practice of amputation for this disease has I believe been almost universally discouraged from the belief that it is generally followed by sloughing of the stump. I wish to urge that this occurs only when the part is removed too near to the disease. If done low down then I fully admit that the condition of the vessels will rarely be found to be such as to admit of repair, and that gangrene of the stump will usually occur immediately, and place the patient's life in much more danger than before.

As regards these operations I have had but little experience, having long believed that they were most unadvisable. The few trials that I have made have fully confirmed this opinion, and I would abstain as scrupulously

in senile gangrene as in frost-bite, from any interference with the knife near to the dying part. If, however, we go much higher up and amputate through parts which are still well supplied with blood the results are quite different.

By operations of this kind I believe I have repeatedly saved the patient's life. By "amputation high up," I mean in the case of gangrene of the foot, above the knee, and in that of the hand at or near the shoulder-joint. By senile gangrene I mean chiefly gangrene in connection with calcification of arteries, but I wish to be understood to include all cognate conditions, that is, all in which the death of the part occurs in connection with slowly progressive interference with the supply of blood. In the strictly senile forms this interference is usually greatest in the distal part of the arterial system, and it is of a nature to be steadily on the increase. Hence the hopelessness of improvement and the great danger of advance. It is impossible that any collateral circulation can be established.

In cases of gangrene from occlusion of the main trunk without disease of the smaller branches, as for instance from embolism or from ligature, the conditions are different. Here there is hope that, when the collateral circulation shall have become established, repair will take place with exfoliation of the sphacelated parts. Yet there are some cases, even of this kind, for which I think high amputation may become advisable. Certainly, amputation near to the gangrenous part should always be avoided unless, indeed, when after repair has considerably advanced there is proof of well-restored circulation. Cases of frost-gangrene stand in some sense midway between these two classes; they are examples of gangrene from distal suspension of circulation as in the senile cases, but the cause is not, as in the latter, a permanent one. There is hope of restoration of the supply of blood. Nevertheless, all authorities are, I think, agreed that this restoration is never sufficiently perfect to make it wise to attempt the removal of the affected parts,

and that it is necessary to leave this almost absolutely to natural processes.

Before proceeding to narrate my cases it will be well that I should here introduce a brief summary of what has been done by others in the same direction.

The proposal to amputate high up in cases of senile gangrene is not absolutely novel. In fact a discussion which bore closely upon it took place in this room more than thirty years ago. On that occasion the late Mr. Garlike of Rickmansworth brought forward a case[1] in which he had amputated in the upper part of the thigh, in a man aged 69, who suffered from senile gangrene in his foot. There was ossification of the femoral artery, and there could be no doubt as to the real nature of the gangrene. The case, however, did not imply any proposal to amputate high up as the best treatment for gangrene of this kind, since it was only determined on after suppurative destruction of the knee-joint, when there was no longer any choice as to the site of the operation, and could be but little difference of opinion as to its absolute necessity.

The case, notwithstanding, is a good example of complete recovery under the somewhat desperate conditions referred to.

In the discussion which followed, Mr. John Adams, my late colleague at the London Hospital, mentioned a case of recovery after amputation for senile gangrene, but the precise part at which the amputation was done is not specified. Mr. Fergusson mentioned that he had operated in a single case, but without success, and spoke with decided disparagement of the practice. To Mr. James, of Exeter, belongs, I believe, the credit of having definitely proposed the line of practice which I shall endeavour to support. Indeed, I believe in one case that he actually did what I only proposed to do, but was not allowed by my patient to carry out, viz. to amputate both the lower extremities. I cannot find that any surgeon, writing since this discussion took place, has recom-

[1] 'Medical Times and Gazette,' 1853, vol. i.

mended the practice, or even thought it worthy of serious consideration.

Even before the date referred to, Mr. Langstaffe and Mr. Guthrie had each of them in single cases amputated above the knee for senile gangrene of the foot. Mr. Langstaffe's patient died in consequence of the hæmorrhage within twenty-four hours, but in Mr. Guthrie's case the stump was almost healed when death from exhaustion occurred. Mr. Mott had, I believe, also done a single operation of the same kind.

Thanks to modern improvements in the details of operations and the management of wounds, the surgeon of to-day approaches the subject with great advantages.

As regards the risk of hæmorrhage during the operation the use of Esmarch's bandage unquestionably gives much real assistance. The calcified arteries, even those of the smallest size, are very easily found, and if tied with their accompanying veins in one loop and with a moderately thick ligature, I believe no difficulty will be found in making them secure.

Some years ago I had a case in the hospital in which the conditions causing the gangrene were complicated and in which the safety and advantages of high amputation were very strikingly exemplified. The patient was a strong-looking man of not more than thirty who had served as a soldier in Canada. There he had suffered from frost-bite of the left foot. It was not, however, followed by immediate gangrene, and a year later he came into the London Hospital with an ulcer on the foot which had destroyed several toes, which would not heal, and which every now and then showed a tendency to spread at its edges by fresh sloughing.

After he had been some time under observation my colleague, Mr. Little, during the summer vacation amputated through the tarsus. When I resumed duty I found the face of the stump gangrenous and the condition of things worse than before. It was now discovered that the femoral artery in the thigh was obliterated. I decided to

amputate again and to do what I then considered a high operation. The leg from the ankle upwards was perfectly healthy, and I amputated just below the knee. The result, however, was that the gangrene attacked the stump, and this time it was an acute form and rapidly spread upwards. The man became exceedingly ill, sick, and delirious, and in this condition I amputated again high up in the thigh and with the most happy results. All the septicæmic symptoms at once subsided, and the stump subsequently healed perfectly.

It occurs to me as very possible that in this case the occlusion of the artery may have been the unsuspected cause of spreading gangrene which took place soon after the frost-bite. My next case seems to be an important item of evidence in this direction.

A man, æt. about 65, received a slight burn in the left hand. The result was spreading gangrene of the fingers, for which he came into the London Hospital. A large part of the hand was then black and sphacelated. We found that he had no pulse at the wrist and that his brachial artery was a solid thick cord. In the axillary, pulsation could be felt. During the next ten days the occlusion extended higher up and involved the axillary. The old man was very ill and clearly about to die if not relieved. I amputated just below the shoulder-joint and found the main artery completely plugged by a dense firm clot. The smaller branches were, however, pervious and bled freely. We had some difficulty with the wound, the edges of which sloughed a little. Ultimately, however, he recovered perfectly, In this case the arteries were not calcified, but very greatly enlarged and thickened. In the opposite limb the brachial artery was visible through the skin as a sinuous pulsating cord down the upper arm and in front of the elbow.

Encouraged by the result in this case, and about the same time, I amputated above the knee for an old gentleman living in Camberwell, but unsuccessfully. He sank within a week with gangrene of the stump. In this

instance the operation was done under most unfavorable circumstances and as a last chance. The patient was stout, in bad health, and the gangrene extensive and advancing. It was of the moist form, and the conditions were so urgent that I did the operation within a day or two of my first seeing the patient.

My next case was one of an ordinary form of senile gangrene of the right foot in a thin old man, æt. 72, in the London Hospital. He suffered also from prostatic retention of urine. Amputation above the knee was followed by good results so far as relief of pain and almost complete healing of the stump were concerned. The patient lived about five months afterwards, but never left his bed. He died ultimately from exhaustion by various causes, and with a gangrenous patch on the other foot. His arteries were very extensively calcareous.

In a fifth case I amputated for spreading senile gangrene of the left foot in an old woman, aged 75. She was suffering severely from the pain and irritation; was almost constantly delirious, and certainly not likely to live more than a week when the operation was done. She was, however, fortunately very thin. After the amputation she did well without an interruption. Her stump healed without a drop of pus and became perfectly sound. Her circulation was so feeble that we had great difficulty in preventing gangrene in the toes of the other foot, and had to keep it wrapped constantly in cotton wool. She returned home about a month after the amputation in tolerable health, but with the other foot still in a condition to cause great anxiety. I had advised her to have that limb removed also, as the best means of making the conclusion of her life comfortable and enabling her to leave her bed. It seemed certain that the toes would at once pass into gangrene if she were to sit up. My advice on this point was, however, declined by her friends. The removal of both lower extremities seemed to them a mutilation too formidable to be considered, and they took her out of the hospital.

The last case which I shall mention is one of unusual interest. The gangrene was senile in the sense that it was caused by calcareous arteries, but the patient was not advanced in years. He was much out of health from other causes, and the gangrenous process was preceded and attended by inflammatory action.

I attended Mr. H— in consultation with Mr. Linton Brunton, of Limehouse, whose relative he was. Mr. H— was only forty-eight, but he had worked hard and lived freely, and although not showing other signs of old age his arterial system had become most extensively calcareous. His pulse at the wrist was most feeble, so much so that frequently for days together it was scarcely perceptible. He was liable to become blue in the face on exposure to cold air, and suffered much from cold extremities. He had once at a railway station fallen unconscious, probably from cardiac syncope, and once or twice after mental excitement had been so prostrate that he was only kept alive by the freest use of brandy. Such being the state of his circulation, he was exposed one snowy day in 1880 to cold, and came home with his feet much chilled. Bullæ formed on his left foot and were followed by gangrenous patches of the skin and much swelling. The bones seemed to suffer more extensively than the skin, and abscesses subsequently formed about them. This might perhaps be explained to some extent by the fact that there was always a certain quantity of sugar in his urine. During the two months that I watched the case Mr. Brunton and I removed piecemeal many of the digital phalanges. Gradually the disease spread up the foot; abscesses with gangrenous patches of skin formed, and the ankle-joint became affected.

We obtained the advantage of a consultation with Dr. Mahomed on December 24th, 1880, concerning the state of the heart, &c. Dr. Mahomed used the sphygmograph and gave me a detailed written report. It was to the effect that there was no proof of arterial disease in the upper extremities, nor any of valvular disease

of the heart; the latter organ was believed to be feeble, possibly dilated, and probably fatty. The pulse was described as "extremely small, non-persistent, readily compressed, short; artery not thickened." The urine was found to contain a large amount of sugar and also a considerable quantity of albumen (about one sixteenth). It had a specific gravity of 1033. Mr. Linton Brunton had often before demonstrated the presence of sugar, but we had never found albumen, nor was it found subsequently.

Mr. H— had been most of this time in a condition of very urgent illness; often vomiting everything for days together, and not in the least expected to live. Towards the end of February his general condition was a little better than it had been, but as the foot got worse and worse we decided to amputate at all hazards. This was done above the knee on February 27th, 1881. The elastic bandage and strap were used, and Lister's precautions fully carried out. When adapting the flaps the femoral artery was observed to project between them like a small bone, and I was obliged to forcibly bend it back. It had been cut in the flaps much longer than the bone, and I feared that we should have trouble with it during the healing of the stump. None such, however, occurred; both the bone and it proved to be sufficiently covered, and the healing was immediate and without suppuration. It is now about three years since this amputation was performed, and the patient has remained well, wears an artificial limb, and gets about almost as well as formerly. His diabetes has for some time disappeared. He has lived more carefully, but has been much troubled by ulcerations on the other foot.

These cases do not include quite the whole of my experience respecting these operations. In a case under one of my colleagues in which, about six months ago, I had pleasure in giving my vote in favour of amputation, the result was, I believe, most satisfactory, and my friends have informed me of several others.

The facts adduced, I think, justify the belief that

amputation through the lower third of the thigh is not a very dangerous operation even in advanced years and with most extensive calcareous degeneration of the arteries. I have never in a single instance had any trouble with secondary hæmorrhage nor encountered any difficulty in securing the vessels at the time of operation. I have always tied with stout catgut or carbolised silk, and have taken up the tissues surrounding the artery freely. The smaller vessels have been twisted, but I have never trusted to this for the main trunk. If it be admitted that the operation is not in itself dangerous, then I think there can be no hesitation in believing that it offers a most acceptable alternative to the miseries of death from slowly advancing gangrene.

ADDENDUM TO THIRD COMMUNICATION

ON

ARTIFICIAL RESPIRATION IN STILL-BORN CHILDREN.

(MEDIASTINAL EMPHYSEMA AND PNEUMOTHORAX IN CONNECTION WITH TRACHEOTOMY.

AN EXPERIMENTAL INQUIRY.)

BY

FRANCIS HENRY CHAMPNEYS, M.A., M.B. Oxon., F.R.C.P.,

ASSISTANT LECTURER ON MIDWIFERY, AND ASSISTANT OBSTETRIC PHYSICIAN TO ST. GEORGE'S HOSPITAL.

Received December 11th—Read December 11th, 1883.

In vol. lxv of the 'Medico-Chirurgical Transactions,' 1882, p. 81, I wrote as follows:

"I have examined the post-mortem records of the Hospital for Sick Children since 1860, and find records of eighty-two cases of autopsies after tracheotomy for various causes.

"Mediastinal emphysema is recorded in five cases, or 6·09 per cent. In no case is pneumothorax noted. . . . I do not imagine that these numbers represent the actual facts; indeed, in the cases in which mediastinal emphysema is recorded, it is almost invariably mentioned by the way as if an unimportant fact; and one recent case was only recovered from oblivion by personal inquiries.

"This is not to be wondered at, considering that, as far as I have been able to discover, the matter is not even *hinted at* in any of the books dealing with tracheotomy; indeed, the following passage is the only mention of the subject which I have been able to find." (Then follows a quotation from Wilks and Moxon, 'Lectures on Pathological Anatomy,' second edition, 1875, p. 308.)

On November 15th, 1883, I received the following statement from Dr. Angel Money, Registrar of the Hospital for Sick Children:

"Since the publication of the above paper there have been 28 cases in which an autopsy was made after tracheotomy; of these 14 were males, 14 females.

"In all cases the examination was made under water.

"In 16 cases out of the 28 (8 males and 8 females), emphysema of the mediastinum was found.

"In 2 of these cases pneumothorax was also found. It was found in no case without emphysema of the mediastinum. The amount of emphysema of the mediastinum was greatest when pneumothorax existed also.

"In many, if not all, cases artificial respiration had been performed."

It will be seen that the occurrence of emphysema was noted in 5 cases out of 82, or 6 per cent. of those cases which ended fatally after tracheotomy in twenty-one years before the publication of the paper above referred to; and in 16 cases out of 28, or 57 per cent. in the two years following its publication.

Pneumothorax was not noted in a single one of the 82 cases occurring in the twenty-one years previous to the paper, but has been noted twice in 28 cases occurring in the two years following its publication.

These facts speak for themselves.

It seems to be evident that emphysema of the anterior mediastinum is a frequent occurrence in fatal cases of tracheotomy.

What its frequency is in cases which do not end fatally cannot be deduced from the above facts; but the occurrence

of mediastinal emphysema can hardly be a matter of indifference, and pneumothorax must be a very serious complication.

It is possible that the bubbles of air in the mediastinum may produce pneumothorax by being ruptured into the pleura by pressure on the sternum and thorax. Such pressure usually (and quite rightly) forms part of the means of recovery where artificial respiration is performed.

It is strange that the occurrence of both complications should have been (but for the casual allusion referred to) completely overlooked until the experiments which I have had the honour of communicating to the Society.

FOURTH COMMUNICATION

ON

ARTIFICIAL RESPIRATION IN STILL-BORN CHILDREN.

CERTAIN MINOR POINTS.

AN EXPERIMENTAL INQUIRY.

BY

FRANCIS HENRY CHAMPNEYS, M.A., M.B. Oxon., F.R.C.P.,

ASSISTANT LECTURER ON MIDWIFERY AND ASSISTANT OBSTETRIC PHYSICIAN TO ST. GEORGE'S HOSPITAL.

Received December 11th, 1883—Read March 11th, 1884.

The following communication concerns certain minor details in the mode of performing artificial respiration in stillborn children, which it has been thought well to test by experiment.

i. It was decided to test the effect produced by the presence of air in the abdominal viscera on the amount of air capable of being drawn into the lungs by the manipulative methods.

Experiment 1.—In a female child ('Med.-Chir. Trans.,' vol. lxiv, 1881, p. 71, Exp. 19, (L.)), in which the manipulative methods (Silvester, Pacini, Bain, Schultze) succeeded in effecting considerable inspiration, a cannula was passed through the abdominal walls at the navel, and

tied in; attached to the cannula was an india-rubber tube with a clip, so that the abdominal cavity could be inflated and the air let out.

The quantity of air inspired by these methods was noted.

It was found that moderate inflation of the abdominal cavity produced no effect on the amount of air changed by Silvester's method, and its modifications by Pacini and Bain. When the abdomen was very tensely inflated, the amount of air inspired was very slightly diminished.

Experiment 2.—In a male child (see experiments above quoted, No. 20 (M), p. 73), the same method of experiment was adopted.

(*a*) The *Pacini-Bain* modification of Silvester's method was repeated six times.

In each case 6 inches (*i. e.* 6 inches on the water manometer) was indicated as the inspiratory value.

The abdomen was then inflated as tightly as possible, and the same method repeated six times.

In each case 6 inches was indicated as the inspiratory value.

(*b*) *Schultze's* method gave 7 inches as the inspiratory value on six repetitions.

The abdomen was then inflated as tightly as possible, and the same method repeated six times.

In each case 7 inches was indicated as the inspiratory value.

Remarks.—The question here was not whether air in the stomach and abdominal viscera impedes respiration when naturally conducted, but whether it impedes *artificial respiration.* These experiments answer the question decidedly in the negative.

The very slight difference noticed in Experiment 1 was probably due to the impeded movements of the thoracic walls, produced by great distension of the abdominal walls.

It must of course be remembered (loc. cit., vol. lxiv, p. 79), that: "This group of manipulations (Silvester's and its modifications), which produces the greatest amount of

ventilation, in no way resembles the normal respiration of a child, which is almost purely diaphragmatic;" and that the presence of air in the abdominal viscera would certainly impede the descent of the diaphragm in a child breathing normally. This, however, is not the question, which really concerns the resuscitation of a stillborn child by artificial respiration.

It must also be remembered that nothing is easier than to empty such a child's stomach of air by gentle pressure.

Conclusion.—The presence of air in the abdomen in no way impedes the ventilation of the lungs by artificial respiration.

ii. It was next determined to test simple methods usually recommended to prevent the entrance of air into the stomach in mouth-to-mouth inflation.

A. It has been recommended to press the larynx, and especially the cricoid cartilage, against the vertebræ, with the idea that the complete cartilaginous ring of the cricoid cartilage will be strong enough to compress the œsophagus without itself collapsing.

This method has been recommended by *Paul Scheel* in 1800, *Herholdt* in 1803, *Olshausen* in 1864, and also by the Committee of the Royal Medical and Chirurgical Society in 1862, who declared that it was competent to effect its object; this, however, applied to adults.

B. It has also been recommended to bend the head strongly backwards, apparently with the idea that the soft œsophagus, lying posteriorly, might become bent in such a way as to be impervious, while the trachea provided with its rings would remain patent.

These points were tested in two experiments.

Experiment 3.—A tube was connected with the pylorus and placed under water, so that bubbles might announce the repletion of the stomach with air. The trachea was connected with a manometer.

A. Mouth-to-mouth inflation was then tried, pressure being put on the cricoid. This was repeated many times.

It was found only occasionally possible to put pressure on the cricoid so as to occlude the œsophagus, without also occluding the trachea.

B. Bending the head back was found to produce no effect in preventing the entrance of air into the œsophagus.

Experiment 4.—A similar method was adopted.

A. Pressure on the cricoid cartilage occluded either œsophagus and trachea or neither. No graduation of the pressure on the cricoid was able to effect a closure of the œsophagus alone.

B. Bending the head back produced no effect.

Conclusions.—Pressure on the cricoid and bending the head back are powerless to prevent air from passing into the stomach if the lungs are inflated.

iii. The next experiment concerned the patency of the upper part of the air passages.

Howard had already shown that the common measure of pulling the tongue forward with forceps is useless, as it does not affect the structures at the base of the tongue, and does not raise the epiglottis.

His manœuvre of tilting up the chin with the mouth closed, however useful in adults, permitting respiration solely through the nose as it does, cannot be of the same use in stillborn children, whose pharynx and air passages are filled with mucus, and in whom the removal of this mucus is one of the great desiderata.

It had also frequently been recommended to lay the head over a table, to allow the head to fall back, away from the lower jaw and the structures connected with it, in order to render the mouth and upper air-passages patent.

It was decided to test this by the frozen section of a fœtus in this position.

Experiment 5.—A stillborn full-time fœtus which had never breathed was consequently frozen in the above position, its head being thrown as far back as it would go. It was then cut in a sagittal plane.

I have to thank Dr. Garson, of the Royal College of Surgeons, for this operation. His saw passed almost mathematically through the middle line of both the front and back of the body.

The result showed that the soft palate lay against the back of the tongue over a large extent, and did not fall away from it.

Conclusion.—Hanging the head backwards over the edge of a table does not provide for the patency of the upper air-passages.

This accords with my own clinical experience. The greatest difficulty occurs in children in the pale stage of asphyxia and probably depends on inaction of the divaricators of the glottis, which are flabby and useless, like all the other muscles during this stage.

This question of the patency of the upper air-passages is often the great difficulty to overcome. It frequently happens (especially in premature children, but by no means exclusively in them) that Silvester's method and its modifications entirely fail from this cause to introduce any air into the lungs. The difficulty is chiefly met with in children in the stage of pale (flabby) asphyxia, and probably for the reason mentioned above.

The method of Schultze certainly succeeds in introducing air under these circumstances in many cases in which Silvester's method and its modifications have failed. The reason of this is not at present clear to me, but I have observed the fact too often to be mistaken.

I have adopted the plan (which I have not seen advocated) of introducing a catheter into the trachea and leaving it there during the respiratory manipulations, and with satisfactory results.

While in the trachea it serves for various purposes, the principal one of which is the removal of mucus, but not in the manner usually recommended. It is advantageous to secure the catheter at the proper length within the trachea, so that it may not slip too far in, or slip out, if the Schultze method is adopted.

I have measured the length along a catheter from the lips to the glottis, and find it 2½ inches in a fœtus.

A catheter which measures 3½ inches from the lips of the fœtus is within the glottis, but above the bifurcation of the trachea.

This then is the proper length, and it is easy to secure the catheter by a soft bandage or by a piece of elastic passed round the head, with which there is no fear of the catheter slipping out or too far in.

The removal of the mucus is best effected in the following manner, and if used should precede all attempts at respiration, in order that the mucus, meconium, and other matters found in the child's mouth may not be inhaled.

1. Lay the child on its back, with the head hanging over the edge of the table, and a little lower than the rest of its body.

2. Wipe out the mouth with a soft handkerchief.

3. Press the thorax gently with one hand, stroking the trachea upwards with the other, and retain the finger at the top of the trachea until the next manœuvre is complete. (The mucus will gravitate towards the posterior nares.)

4. Put a handkerchief over the child's mouth, blow gently, and the mucus will be blown out of the nostrils (but not into the operator's face).

5. If necessary, introduce a catheter 3½ inches from the lips and secure it.

6. Press the thorax gently with one hand (to prevent the entrance of air), and *blow* through the catheter. The opening being low down in the trachea, the air and mucus with it being unable to pass into the lungs on account of its compression by the hand, will rush up through the glottis, and the mucus will be blown into the pharynx. This can be repeated as often as necessary, the general tendency of fluids in the air-tubes being to ascend during respiration, whether natural or artificial, towards the mouth.

This manœuvre is more efficient and far pleasanter than the suction usually recommended; it has answered well in practice.

During artificial respiration fluid will often pour from the catheter, especially if it is held over the edge of the table like a syphon.

N.B.—The plan of *holding the nostrils* during inflation of the lungs is altogether to be condemned; it retains mucus which might be expelled, and the nostrils are a *valuable safety-valve* guarding the lungs from over-distension.

iv. I have thought it worth while to describe some of the progressive signs of returning life in a case in which I had good opportunities of observing them.

A very large male child was delivered on April 14, 1878, in a state of pale (flabby) asphyxia after turning, indicated by a hand presentation. There was exomphalos, and mouth-to-mouth inflation showed the air to pass freely into the intestines, which protruded considerably.

The child was born at 3.20 p.m., the only sign of life being a very feeble and slow beating of the heart.

Artificial respiration was begun by mouth-to-mouth inflation, and continued by the methods of Silvester and Schultze, assisted by alternate hot and cold baths. Under this treatment the heart's action became stronger and more rapid.

The first breath was drawn at 3.50 p.m., but breathing was not spontaneous till 4.30 p.m. and was then very feeble. When it was at length established, expiration was assisted by pressure on the thorax and abdomen.

The breaths were not equally drawn, but about every third breath was deeper, the intermediate breaths being light and shallow.

Breathing was fairly established at 5.15 p.m.

The first sign of muscular action was seen about 4.45 p.m., and consisted in a quivering of the tongue; then the alæ of the nose dilated, then the mouth quivered. About this time the expirations began to be tremulous as in crying. About 5 p.m. there was "sobbing," *i.e.* the diaphragm produced irregular inspirations without consentaneous opening of the glottis.

General Conclusions.

1. The presence of air in the abdominal viscera in no way impedes the ventilation of the lungs by artificial respiration.

2. Pressure on the cricoid cartilage, and bending the head back are powerless to prevent the passage of air into the stomach, during mouth-to-mouth inflation.

3. Hanging the head backwards over the edge of a table does not provide for the patency of the upper air-passages.

A plan of providing for the patency of the air-passages and the removal of mucus is here related in detail.

Notes of the signs of returning life in a deeply asphyxiated child are given from a careful observation.

Works quoted.

Scheel (*Paul*). Ueber Beschaffenheit und Nutzen des Fruchtwassers in der Luftröhre der menschlichen Früchte. Aus dem Latein. mit Anmerkungen. Erlangen, 1800, S. 53.

Herholdt (*Johann Daniel*). Commentation über das Leben, vorzüglich der Frucht im Menschen, und über den Tod unter der Geburt. Kopenhagen, 1803, S. 164.

Report of the Committee appointed by the Royal Medical and Chirurgical Society to investigate the subject of Suspended Animation. Med.-Chir. Trans., vol. xlv, 1862, p. 490.

Olshausen (*R*). Die Behandlung scheintodter Neugeborener durch künstliche Respiration. Deutsche Klinik, 1864, Band xvi, S. 346, 357, 365.

Howard (*Benjamin*). Observations on the Upper Air-passages in the Anæsthetic State. Lancet, 1880, vol. i, p. 796.

Champneys (*F. H.*). Artificial Respiration in Stillborn Children. Med.-Chir. Trans., vol. lxiv, 1881, p. 41.

THE DIRECT TREATMENT

OF

PSOAS ABSCESS WITH CARIES OF THE SPINE.

BY

FREDERICK TREVES, F.R.C.S.,

ASSISTANT SURGEON TO, AND LECTURER ON ANATOMY AT, THE LONDON HOSPITAL.

(Received October 15th, 1883—Read January 8th, 1884.)

Among inflammatory affections of bone spinal caries must rank as one of the most serious, yet its gravity depends upon no special virulence of character, upon no unusual activity of progress. The caries that involves the vertebræ is not an isolated and peculiar variety. It is usually of the kind known as strumous, or better, as osteo-myelitis granulosa. It may be of the simple species that follows upon injury, or, in rarer instances, of the form that is dependent upon syphilis. A caries precisely similar to it may be met with in the foot or hand; and yet an amount of disease may prove fatal in the spine that in the os calcis would form but a comparatively insignificant ailment. Its gravity can hardly be said to depend even upon the proximity of the malady to the spinal cord. Bone disease in the upper cervical region is certainly very often fatal by

the mischief it may induce in the medulla spinalis, but elsewhere in the column it must be owned that the fatal issue is comparatively seldom due wholly to a central nerve complication. It cannot even be said that in the vertebræ caries meets with a bone tissue that presents any facilities for its development that are not possessed by many other bones in the body. In actual fact the carpus and tarsus present advantages for the rapid progress of caries that are greatly in excess of any possessed by the spinal column.

It is needless to say that the seriousness of Pott's disease depends mainly upon the depth at which the morbid centre is placed; upon its inaccessibility to methods of treatment that are successful in other forms of caries, and upon the tedious, laborious, and destructive processes that are necessary for the evacuation of its morbid products.

When caries attacks the spine suppuration as a rule results, as it does in carious processes elsewhere. The abscess formed has, however, not attained great size before it finds itself hemmed in on all sides. All ready points of exit for its escape are closed, and to find an issue upon the surface of the body it has to take a long and devious course. When the malady involves the lower segments of the column the pus usually follows the psoas muscle and so makes its way to the thigh. Or it may fall short of the thigh and wander into the pelvis or force its way among the connective tissue planes of the lumbar region. Wherever it goes it leaves behind it a tract of destruction and adds to the original evils of the disease a grave and terrible complication. But even when the products of suppuration have been evacuated the disease is still far from cure. There is an immense abscess cavity that must be closed and a vast amount of dead and dying bone in the depths of the body that must be more or less completely eliminated. Nature's methods of effecting this removal are very slow. The products of the disease are carried away particle by particle and grain

by grain, and it not infrequently happens that the patient dies worn out before the tedious process is completed.

Now take for the sake of comparison a case of caries involving a superficial bone. Suppose, for example, that a patient is attacked with this disease in the vicinity of the wrist. The parts involved can at once be placed at rest. Any pus that may form can be immediately evacuated. Necrotic fragments or portions of disintegrated bone can be readily removed, and the progress of the disease, with more or less success, arrested.

Surgeons having under their care cases of Pott's disease must have been impressed with the good that might accrue if only that malady could be made amenable to such methods of treatment as are usually adopted in the more superficial forms of caries; but I am not aware that this impression has been carried into actual practice.

In the present paper I venture to submit to the notice of this Society an operation for the more direct relief of spinal caries when associated with abscess and when occurring in a certain segment of the column. I might say at once that the procedure is concerned only with the disease when it involves the lumbar and, with some reservation, the twelfth dorsal vertebræ. It consists simply in cutting down upon the bodies of those vertebræ through an incision made in the loin. By means of this incision the diseased area can be more or less directly exposed, suppurative collections can be opened at their point of origin and evacuated by the shortest possible route, portions of necrosed bone can be removed when such exist, and the diseased district subjected to the same methods of treatment that are commonly applied to the more superficial forms of caries.

I am enabled to give an account of three patients upon whom I performed this operation, and from one of whom I removed a large sequestrum involving a great part of the body of the first lumbar vertebra.

The details of the operation as it may be performed on the cadaver are as follows. In the description given I have

presumed that the surgeon intends to cut down upon the second or third lumbar vertebra. If a higher or a lower vertebra require to be exposed some slight and obvious modifications as regards position of incision, &c., would have to be introduced. These, however, need no detailed notice. The patient's loin having been exposed a vertical incision some two and a half inches in length is made through the integuments. The centre of this cut should lie about midway between the crest of the ilium and the last rib, and the cut should be so placed as to correspond to a vertical line parallel with and to the vertebral side of the outer border of the erector spinæ. I find that the average width of the erector spinæ in this situation is, in the adult, from two and three quarters to three inches. The incision, therefore, should be situated about two and a half inches from the lumbar spinous processes. After cutting through the superficial fascia the dense aponeurosis is exposed that covers the posterior surface of the erector spinæ and which is variously known as the superficial layer of the lumbar fascia, as the aponeurosis of the latissimus dorsi and serratus posticus inferior muscles, and as the inferior part of the vertebral aponeurosis. The part of this layer exposed in the lower half of the incision is wholly tendinous, but from that seen in the upper half of the cut arise some of the fibres of the latissimus dorsi. These fibres are thin and pass from below obliquely upwards and outwards. The dense aponeurosis with its attached muscular fibres having been divided in the full length of the incision the erector spinæ is exposed. This muscle is at once recognised by the vertical direction of its fibres. The outer border of the muscle should now be sought for and the whole mass drawn by means of retractors, as far as possible towards the middle line of the back. In this way the anterior part of the sheath of the muscle, known as the middle layer of the fascia lumborum, is readily exposed. Neither in front nor behind has the erector spinæ any direct adhesion to its sheath at this part.

The anterior layer of the sheath, as now exposed, is seen to be made up of dense white glistening fibres which are all more or less transverse in direction. Through this sheath the transverse processes of the lumbar vertebræ should be sought for. The longest and most conspicuous process is that belonging to the third vertebra. It is readily felt. The erector muscle having been drawn as far as possible towards the middle line, the anterior layer of its sheath must be divided vertically as near to the transverse processes as convenient. By this incision the quadratus lumborum muscle is exposed. The muscle as here seen is very thin. It is composed of muscular fibres which run from above obliquely downwards and outwards. Between the fibres are tendinous bundles which spring from the tips of the transverse processes. The muscle should be divided close to the extremity of a transverse process and the incision cautiously enlarged until the muscle is divided to the full extent of the skin wound. It is at this stage that there is danger of wounding the abdominal branches of the lumbar arteries. The inner edge of the quadratus is overlapped by the psoas muscle, so that when the former is divided the latter is exposed. The psoas fibres, as now seen, take about the same direction as the posterior fibres of the quadratus, *i.e.* run downwards and outwards. The interval between the two muscles is marked by a thin but distinct layer of fascia, known as the anterior lamella of the fascia lumborum. Some of the tendinous fibres of the psoas having been divided close to a transverse process the finger is introduced beneath the muscle and gently insinuated along the process until the anterior aspect of the bodies of the vertebræ is reached. The incision in the psoas can be enlarged to any extent.

If the patient were stout or very muscular the length of the skin wound would have to be increased, or a transverse cut might be made into the erector spinæ to allow of its more effectual retraction.

With common care there should be no danger of opening up the subperitoneal connective tissue, much less of

wounding the peritoneum. All risk on this score will be avoided by making the incision in the quadratus as near to the transverse processes as possible.

Great care must be taken not to wound the lumbar arteries. The abdominal branches of these vessels run for the most part behind the quadratus lumborum. That, however, from the first vessel runs in front and not infrequently those from one or two of the lower arteries follow its example. These vessels may be of large size, often as large as the lingual. They may be avoided as well as the trunks from which they arise by keeping close to a transverse process. The main vessel curves around the spine between the transverse processes and between these processes also the division of the artery occurs. If, therefore, the rule be observed of always reaching the spine along a transverse process, the lumbar arteries and their abdominal branches need be exposed to no risk.

In actual practice the operation is much simpler than may appear from this description. The patients are usually young and thin—often very thin. If the disease have lasted for any length of time the muscles about the part are found to be atrophied, and if any moderate deformity exist it serves to render the morbid region more easy of access. The moment, moreover, that the quadratus is incised the psoas is reached, and here will in all probability be found an abscess cavity which will immediately conduct the surgeon's finger to the seat of the disease. As to which loin should be selected—the right or the left—it matters little. The operation can be somewhat more conveniently performed upon the right side, while upon the left it is that the risk of damaging the peritoneum by an accidental slip is reduced to a minimum.

I might now give an account of the three patients already referred to. They came under my care at the London Hospital while doing vacation work for my colleague Mr. Adams, to whom I am indebted for permission to publish them.

The first patient, a young woman, æt. 21, was admitted on July 1st, 1883. Two years before she had received a violent blow over the lumbar spine. Twelve months before she began to experience pain in this part on movement, which pain had continued more or less until she came into the hospital. Two months before admission a swelling appeared over the right iliac crest and since that time walking had been so painful and difficult that she had to take to her bed. Her state on admission was as follows: She was anæmic and very thin. There was tenderness over the first and second lumbar spines, which was much increased by movement; there was great rigidity of this segment of the spine, but no deformity. A small swelling existed over the right iliac crest, and a large dull and fluctuating swelling in the left iliac region. This latter swelling, which was evidently an immense abscess, could be felt on examination by the rectum and vagina to extend into the pelvis.

The patient's condition a month later, *i.e.* early in August, was as follows. The collection in the left iliac region had increased rapidly and had been aspirated three times. After each tapping it refilled speedily, the amount of pus evacuated on the three occasions being respectively 28, 60, and 30 ounces. Thus no less than three quarts of matter had been discharged in about thirty days. The patient was rapidly emaciating; she had severe night sweats, her temperature ranged from 100 to 104, she was extremely feeble and complained of so much pain in the left side as to require the frequent administration of hypodermic injections of morphia; she had occasional rigors and was often sick. It was evident that this enormous amount of suppuration was kept up by some abiding source of irritation, situated probably in the lumbar spine. The parts had been kept at rest by maintaining the recumbent posture and by restraining movements by means of a long Liston's splint. The patient, however, was not able to endure the confined position for long, and the splint was left off. Under the circumstances, therefore, I resolved to perform the operation above described with

the purpose of hitting the abscess near its point of origin and of removing if possible any carious bone that may be perpetuating the mischief. The operation was performed on August 11th. The incision was made in the left loin. The erector spinæ muscle was found to be much atrophied, and before the quadratus lumborum could be well defined a huge abscess was opened that occupied the substance of the psoas muscle.

Introducing the finger into the abscess cavity the bodies of the lumbar vertebræ were soon reached, and in the substance, or rather in the place, of the first vertebra a large sequestrum was found. This was readily removed. It was situated in a cavity apparently lined by granulations, and after its removal no more dead or exposed bone of any kind could be detected. The operation was conducted under antiseptic precautions, and from the abscess cavity some forty ounces of pus were evacuated. It was evident that the abscess extended far down into the pelvis. A large drainage tube was introduced so as to reach the vertebral column. The sequestrum was composed of a mass of cancellous bone and measured about one inch by half an inch. The improvement in the patient's condition was immediate and very marked. Her temperature sank so as to reach nearly the normal line. She no longer suffered any pain. Her appetite improved and she began to gain flesh rapidly. By September 24th the patient was able to sit up for the greater part of the day, the sinus left by the wound had greatly contracted, and the amount of discharge had become quite insignificant. On the 29th of September the antiseptic dressings were discontinued. The patient is now well. She walks about without inconvenience and is much improved in appearance and in general health. There is no longer any tenderness about the spine, and the wound has quite healed.

The second case is of a different character and serves merely to illustrate the ease with which the lumbar spine can be examined under circumstances of doubt. The

patient was a little girl æt. 12, who was admitted on July 25th, 1883. She had a conspicuous angular curvature in the lumbar region of the spine, the most prominent spinous process being that of the second vertebra. She was an orphan and without friends, so that no account of her past history could be obtained. All that could be ascertained was that the "lump" in the back had been there for some months, that she had had no accident, and had not been laid up. She was pale, thin, and ill-nourished, and in the right groin there was a small, dull, fluctuating swelling painful on pressure. I concluded that this abscess was connected with the mischief in the spine. On August 15th the swelling was aspirated and two ounces of pus evacuated. About a week afterwards pus spontaneously escaped through the trochar puncture and the abscess was then freely opened with the scalpel. It proved to be a small chronic abscess of no great magnitude and apparently was in no way associated with the spinal affection. Its origin, indeed, was probably quite casual.

By the 1st of September the abscess had almost ceased to discharge, but the child had become much worse. Her temperature has been rising for the last two days; she looked ill; she had lost her appetite, and she complained of vague pains about the lower part of her back. There was then no symptom that directed any attention to the lungs. I imagined—erroneously, as the sequel proved—that these evidences of inflammation depended upon some fresh mischief about the lumbar spine, since they were in all points consistent with commencing suppuration. I therefore cut down upon the affected vertebræ from the right loin by the procedure already described. The spine was readily reached, and my examination of it merely revealed great inequality of the bony surfaces and great thickening of the soft parts about the site of the deformity. It was evident that the spinal disease had been long cured and had nothing to do with the child's present symptoms.

The operation was performed antiseptically, and the

wound did well. On the following morning the child was found to be breathing rapidly, and an examination of the bases of the lungs revealed extensive dulness and tubular breathing. The patient remained for some time very ill, and on September 27th evidences of empyema having appeared the chest was tapped and much pus evacuated. Since then the child has done well and has been some time discharged from the hospital. In this case I must confess that the operation was based upon error, and I have to congratulate myself upon the circumstance that the case ended as favorably as it did. It, however, served to show with what comparative ease the lumbar vertebræ can be subjected to examination.

The third patient was a lad æt. 16, who was admitted into the London Hospital on October 19th.

In his case an angular deformity in the lumbar region was noticed two years ago. Some six months ago an abscess was opened in the right groin about one and a half inches below Poupart's ligament. The boy has been under treatment at different hospitals since the incision was made and has been to the seaside. On admission the sinus was discharging very freely, the patient had suffered greatly in health from the continuous drain of pus, and nothing that was done in the way of irrigating the abscess cavity, had any effect upon the amount of the discharge. On October 27th the incision already described was made along the right side of the lumbar spine. The angular deformity was considerable, the most prominent spinous process corresponding to the second lumbar vertebra. This bending of the spine had brought the transverse processes more together, and in order to more readily reach the psoas muscle I removed with bone forceps the transverse process of the third vertebra. No pus had been encountered by the time that the psoas muscle was reached, but upon cutting into that muscle an abscess cavity was opened up from which pus escaped through the wound. I found that the bodies of two of the lumbar vertebræ lay in the abscess cavity, and from the surface

of bone thus exposed I removed with the finger a few minute fragments of carious bone. I believe that no active mischief was progressing in the bones, but rather that their condition at the time of the operation depended upon their having been long kept in contact with retained and offensive pus. The operation was performed antiseptically, but as the whole abscess cavity was rendered septic by the communication of the sinus in the thigh with the air the dressings were soon abandoned. When a few days had elapsed I passed a large drainage-tube in at the sinus in the groin and brought it out at the wound in the back. By this means the whole psoas muscle was drained from one end to the other and the entire abscess cavity could be readily washed out.

By December 3rd the discharge had become much less than it was before the operation, the boy was improved in health, and was free from pain. An examination of the part under chloroform revealed the fact that the bared surfaces of bone were now quite covered over by granulations and no bare bone could be anywhere felt in the depths of the wound.

As the discharge of pus was more free on some days than on others, I imagined that the abscess cavity might be sacculated and that within these sacculi pus might be lodged. To meet this condition it appeared desirable that the cavity of the abscess should be irrigated more frequently than twice a day. By passing a very large fenestrated tube through the length of the psoas muscle, and by making use of a syphon arrangement, a continuous stream of cold water was allowed to run through the abscess cavity from one end to the other. This was kept up for seven days, during which time the boy's temperature remained at or but little above the normal. After the removal of the tube the discharge was found to be greatly diminished. The lad succumbed to chronic lung disease two months after the operation. The autopsy showed that the once carious bone was covered over by healthy granulations, and the once large abscess cavity in

the psoas muscle was reduced to a very narrow tract. (The specimen was exhibited.)

In conclusion I would draw attention to one or two pathological circumstances that more or less directly concern this operation.

In the first place it cannot be said that the lumbar region is so seldom the seat of caries as to render the operation of very limited application. According to Hueter the twelfth dorsal and the first lumbar are the vertebræ that of all others are the most frequently attacked with this disease. The latter of these vertebræ can be reached without difficulty, but the last of the dorsal centra is much less accessible. It may be noted, however, that the psoas fibres reach to the lower border of this vertebra and that the muscle may be made use of to reach the bone. Probably in most cases an abscess would exist, under which circumstances the abscess cavity would form an easy and safe guide to the surface of the vertebra.

In the second place it must be remarked that in the great majority of all cases of spinal caries the disease commences in the superficial parts of the bone, if not actually in the subperiosteal tissue. This being the case, an operation such as has just been described could be effectively carried out at the very earliest stages of the disease, and at a time when the extent of the malady was yet insignificant. Sometimes the disease has commenced in the centre of the bone and even at its posterior surface; but in all such cases there is a tendency for the morbid process to advance towards the anterior aspect of the bone and thus to assume a position in which it would be more amenable to treatment. Probably the least satisfactory cases would be illustrated by those few examples of vertebral caries where the disease has commenced simultaneously in two or more points of the column. Such cases are, however, I believe, very rare.

Thirdly, it must be borne in mind that when much angular deformity exists the space between the last rib

and the iliac crest becomes much encroached upon. Indeed, the rib may touch and even overlap the pelvic bone. Cases, however, where the space available for this operation is seriously narrowed are not common, are mostly found in instances of extensive disease in the dorsal spine, and are, for several reasons, unsuitable for active treatment.

Lastly, I think it may be said in favour of this operation that it directly reaches the seat of disease and renders the affected part amenable to modes of treatment that are commonly adopted in like maladies attacking more superficial bones. It opens any abscess that may exist at the actual seat of disease, and, so long as the patient retains the recumbent posture, at its most dependent spot.

It evacuates the matter by the shortest route, and if applied early enough should prevent the formation of a huge abscess that may burrow into the iliac region, the pelvis or the thigh. I would urge that a psoas abscess when diagnosed, should be opened, other things being equal, at its point of origin from the lumbar spine rather than at a spot quite remote from that point. In suggesting this mode of treatment I am merely advocating for the relief of psoas abscess the same method that is usually adopted in suppurating collections more favorably placed. Moreover, when the whole psoas muscle has been converted into a huge abscess and when the pus is pointing in the thigh or is actually discharging at that spot, I think that by evacuating the matter through an opening in the loin and a counter opening in the thigh and by draining the whole cavity from one end to the other, one is merely carrying out a recognised and successful plan of treatment that has been extensively applied to abscesses elsewhere.

I am well aware, however, that this method of treatment, even if entirely carried out, cannot place spinal caries in the same position as regards opportunity for cure in which surgery has placed carious maladies of surface

bones, until two great obstacles are removed—the difficulty of recognising the disease at its earliest stages and the difficulty of keeping the spine at absolute rest.

RADICAL CURE

OF A

LARGE SPINA BIFIDA IN AN ADULT.

BY

WALTER WHITEHEAD, F.R.C.S.E., F.R.S. Edin.,

SURGEON, MANCHESTER ROYAL INFIRMARY; SENIOR SURGEON, MANCHESTER AND SALFORD LOCK AND SKIN HOSPITAL.

Received January 22nd—Read January 22nd, 1884.

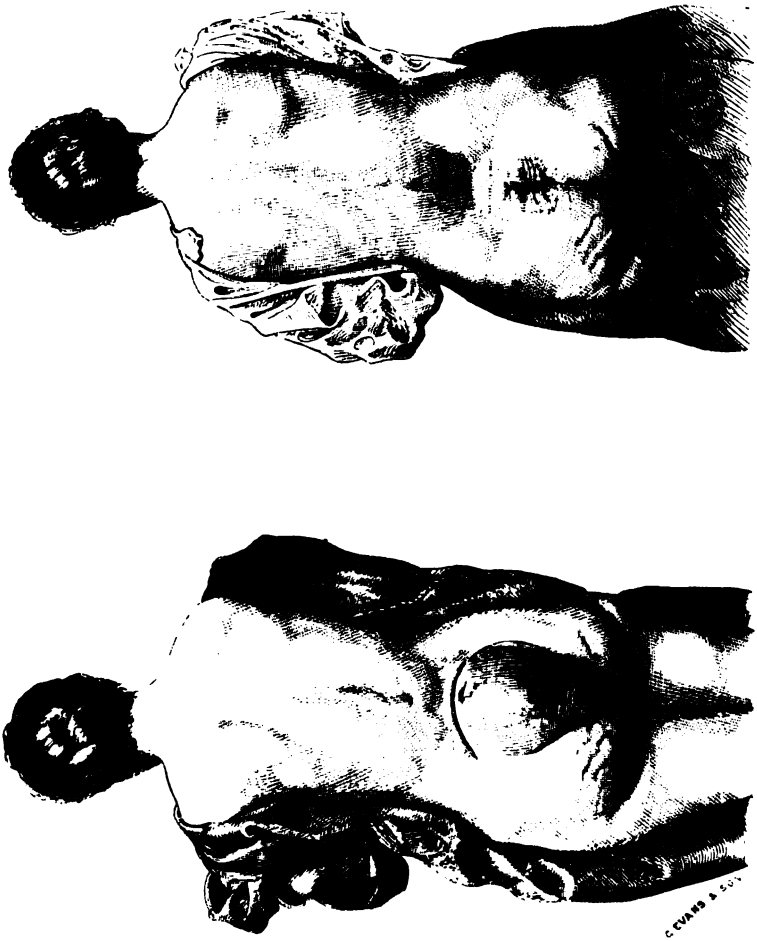
Alice A—, æt. 27, was admitted into the Manchester Royal Infirmary July 11th, 1883, with a large spina bifida situated in the lumbro-sacral region. She described the tumour, which had existed from birth, as having remained about the size of an ordinary hen's egg until she had attained the age of twenty-one, without causing her the slightest inconvenience. When eighteen years old she contracted an unfortunate marriage, which terminated in a complete separation within three years. During this short period she cannot remember any change taking place in the character of the tumour, or that she suffered from any symptoms which she could attribute to its existence. After leaving her husband, her circumstances compelled her to adopt means of earning her own living; and for this purpose she obtained a situation as a "hooker" in a printing warehouse, where she was employed in duties that involved the lifting of heavy goods and other offices

of an arduous nature. From this time she dates, and to the laborious work she ascribes, the first change that took place in the tumour, which had remained for so many years a passive and unobtrusive malformation. For eight years she continued her employment, and during this time the tumour gradually increased in size; and coincident with the enlargement symptoms of a distressing character developed. She commenced to have severe headaches, attended with nausea and vomiting, attacks of vertigo, and transient moments of defective vision, whenever the tumour was subjected to pressure, even that of the clothing. These symptoms were intensified whenever her duties required that she should bend her body. The dimensions of the tumour also commenced to make locomotion difficult and awkward. The size of the tumour was always the greatest in the evening. She followed her employment eight years, when she had to abandon it altogether from a total incapacity to continue it any longer, the intervals between the attacks having become rare and shorter in duration.

When she was admitted into hospital the tumour was sessile, pyriform in shape, of regular outline, and situated over the sacrum. The base was three inches higher than the iliac crest, and the apex extended to within an inch of the tip of the coccyx. The dimensions were:

Circumference . . .	22 inches.
Transverse measurement . .	12 „
Vertical measurement . .	10 „

The appearance of the patient before and after operation is well represented in the woodcuts, page 129. The skin covering the tumour was healthy, though evidently much attenuated; there were several large veins traversing its surface. At the upper border there was a circular somewhat depressed patch of cicatricial tissue suggestive of ulceration having occurred at this point during infant life. The tumour felt tense and elastic, and there was clear evidence of fluctuation. Pressure produced pain in the head and a feeling of tension behind the eyes. By means of transmitted light (the oxyhydrogen lamp being employed) the tumour was shown



to be translucent, and it was easy to define through the breadth of the tumour the contour of a small coin, or the edge of a card placed between the light and the opposite side.

August 17th.—The tumour was tapped with one of Southey's fine trocars, and 12 oz. of fluid withdrawn, but this only slightly diminished the size of the swelling.

The fluid was examined and found to contain $\frac{1}{16}$ albumen, no sugar, and had a sp. gr. of 1025.

Notwithstanding the careful application of collodion to the aperture made by the trocar, the fluid continued to escape for the succeeding twenty-four hours, and on the following day the tumour was found quite small and flabby. Advantage was taken of this opportunity to examine the connection between the sac and the spinal column.

By invaginating the skin, a finger could detect a deficiency of triangular shape corresponding to the last lumbar vertebra and the upper part of the sacrum. The aperture apparently measured about an inch and a half by one inch. The collapse of the tumour was succeeded by six days' constant and violent headache, associated with nausea and vomiting, pains between the shoulders, and a burning sensation in the muscles of the legs and arms. These symptoms continued with unabated violence for six days, when they were promptly arrested by the introduction of an old-fashioned seton behind the neck.

On the 4th October the seton in the neck was discontinued and under the ordinary antiseptic precautions a single horsehair was passed through the tumour, with the intention of testing the effect of gradual drainage. The tumour was covered with gauze dressings, and these were changed whenever it was observed that the fluid had soaked through them. On the third day the horsehair was replaced by a double strand of fine silver wire (No. 27) as the horsehair did not keep the aperture open. On the fourth day the tumour had entirely collapsed for a second time. During the process of drainage, the patient's symptoms assumed an alarming character: violent head-

ache, sense of fatigue in the legs, vertigo, sickness (every meal being rejected), retention of urine. For seventeen days in which the drainage treatment was continued, the catheter had to be constantly used, and the pains controlled as effectually as possible by morphia administered hypodermically. During this time it would be impossible to estimate the quantity of fluid that escaped; however, forty-eight hours was a sufficient time for the tumour to refill. On the 21st October, as the constitutional symptoms continued of a most distressing and alarming character, high temperature (102°), acute pain in the entire length of the spine, spasmodic contraction of the dorsal muscles, and cramp in the muscles of the legs, the actual cautery was freely applied in lines to the entire surface of the tumour, as a powerful and prompt counter-irritant, and the integument was immediately coated with collodion. From this time the patient's general symptoms rapidly subsided, and on the second day she was practically free from any symptom calculated to excite alarm. On the third day, after the application of the cautery, the tumour was aspirated and 3 oz. of clear fluid withdrawn; on the fifth day, 2½ oz. were abstracted and found to be slightly tinged with blood and to contain flakes of lymph. On the twelfth day, 6 oz. of thick pus were withdrawn. On the thirteenth day, chloroform was again administered, and the tumour was laid open by a single incision from the top to the lowest point; a quantity of pus was evacuated. The cavity was loosely packed with iodoform gauze.

December 20th.—Convalescent.

The patient was present at the meeting for the inspection of the Fellows, having made a complete recovery, and suffering in no respect whatever from her previous ailment.

The following appear to be the most obvious conclusions to be drawn from the foregoing case:

1. That the walls of the sac consisted of dura mater and arachnoid matted together; and that it merely contained subarachnoid fluid and possibly some of the posterior nerves, but no portion of the cord itself.

2. That the aperture of communication between the spinal canal and the tumour was small from the first; and that probably from some cause the canal became occluded or reduced to such fine proportions that no material impulse was conveyed by the fluid from the central cavity to the interior of the tumour.

3. That this case may with advantage be studied, in conjunction with that reported by Erichsen in 'Science and Art of Surgery,' 7th edit., vol. ii, p. 290.

4. That the favorable termination of the case must, I presume, be attributed to the antiseptic precautions and the traumatic inflammation induced by the application of the actual cautery, when the plastic character of the inflammation closed the communication.

5. That the result of the case suggests the propriety of testing the efficacy of the actual cautery in other cases of spina bifida, where it may reasonably be supposed that the tumour contains neither the cord nor anterior nerves of the spine.

ON A FORM

OF

ALCOHOLIC SPINAL PARALYSIS.

BY

W. H. BROADBENT, M.D., F.R.C.P.

LECTURER ON MEDICINE AT, AND PHYSICIAN TO, ST. MARY'S HOSPITAL.

(Received December 11th, 1883—Read February 12th, 1884.)

SEVERAL cases of a remarkable form of spinal paralysis have come under my notice at different times, but, with the exception of the first, which occurred in St. Mary's Hospital when I was resident medical officer there, the patients have been seen in consultation once only, and no post-mortem examination has been obtained, so that it has been impossible to give anything like a complete and connected account of the disease. This I am now able to do through the zealous co-operation of Dr. H. F. E. Harrison, of Shepherd's Bush Green, who called me to a case of the kind on the 3rd of April, 1883.

The patient was a gentleman, æt. 42. He was by profession a barrister, but had never practised, having independent means. As a young man he was a cricketer, and had played for his college and in the eleven of his county, but he early developed a liking for wine and gradually became inert and sedentary. In March, 1875, he came under Dr. Harrison's care and was then a confirmed dipso-

maniac. His favourite drink was port, and his habit was to begin drinking about mid-day and to go on more or less all day and through the night. He spent most of his time in bed, reading and drinking, and of late years was seldom seen out of his house except at the University cricket match or on some similar occasion.

It is probable that these habits had an important influence on the form taken by the injurious effects of alcohol. He never had pronounced delirium tremens, but at times complained of wakeful nights and of seeing spectres, and he was easily startled and frightened. For some months his friends had observed a degree of failure of mental power, shown chiefly by his tendency to ask the same question or repeat the same remark over and over again.

The precise date of the access of the paralysis cannot be fixed. He had been suffering much from vomiting and had been in bed for a month; slight weakness of the arms had been noticed for about a fortnight before April 3rd, when I was called in consultation, but it had only interfered with the use of the hands in holding his paper or carrying his glass to his mouth for a day or two.

On examination at this time (April 3rd), the attention was at once called to the hands, which could no longer hold an object, and there was conspicuous dropping of the wrist, especially of the right limb. When the forearm was prone the hand fell by its own weight, and extension was impossible; the grasp also was extremely feeble, partly from loss of opposition to flexion at the wrist by the paralysis of the extensors, but also from weakness of the flexors. Pronation and supination could be effected in a languid way; flexion of the elbow was fairly energetic, and the hand could be carried promptly to the head and behind the neck, although of course it hung loosely at the wrist. The left upper extremity was in a similar condition, but the paralysis of the extensors of the wrist had not gone so far, and the hand could be brought into line with the forearm, although, in the absence of effort, it dropped like the right.

The muscles of the forearm were of average volume, but

soft to the feel; they were very tender when handled. The hands were pale and slightly puffy with a purplish tinge.

The legs were very thin as compared with the arms. They could be raised from the bed, and flexion and extension of all the joints could be performed readily. The patient, however, could not stand alone.

The knee-jerk was entirely absent.

In raising himself into the sitting posture the patient required assistance, and it was evident that the muscles of the trunk were enfeebled. He could sit upright, however, and turn in bed.

As the legs hung down the feet could be seen to swell so that in a few minutes they became distinctly puffy.

There was no paralysis of the face or tongue, and the voice was strong and distinct.

Sensation was apparently unaffected. There was no umbilical reflex, but when the feet were tickled they were quickly withdrawn, and the patient exclaimed that he could not bear it.

The cheeks had a good colour, which, although due to dilated capillaries, gave the patient the appearance of health. The eyes were unduly open, giving him a startled or wild look and the pupils were large, but equal and acting well.

The pulse was regular, but frequent, small, short, and weak; there was no distinct cardiac impulse or apex beat; the first sound was short and fairly loud, the second extremely weak and following the first at an abnormally brief interval. The general effect was a singular accentuation of the first sound, the second following it immediately like a weak echo.

The liver was large, and its margin was felt to be firm and thick; the abdomen presented nothing remarkable.

The urine was of a dark-red (almost brick-red) colour; acid in reaction, with a sp. gr. of 1028, throwing down a copious deposit of red urates; it contained neither albumen nor sugar.

All alcohol was withdrawn; beef-tea and milk were given as food, and Liq. Strychniæ ɱvj was ordered as medicine.

He became rapidly worse, and on April 9th he lay on his back unable to turn in bed or move his body in any way; his arms were lying along the body, he could just raise the right hand from the bed and drag it partly on to the side of the abdomen; the left he could only move slightly but could not lift, being thus more affected than the right. The hands fell by their weight into extension or flexion according to the position of the forearm. From their position, which was somewhat dependent, they were much more puffed and swollen than before, and pink in colour.

The legs could only be freely drawn up and could not be lifted from the bed. There was no knee-jerk or ankle-clonus. Tickling the soles made him cry out and withdraw the feet.

There was no umbilical reflex, and tickling of the flank excited no complaint.

The aspect and expression of the face were more natural than at the previous consultation and the pupils less dilated, perhaps because of the withdrawal of alcohol. The voice was good and articulation quite distinct.

With the general increase of paralysis there was now obvious paralysis of the diaphragm. The patient did not complain of respiratory distress, but the movements of the upper part of the chest were observed to be greatly exaggerated, and both inspiration and expiration were noisy. On examination it was seen that the abdominal respiratory movements were reversed, the epigastrium receded in inspiration and was forced outwards in expiration, showing that the diaphragm did not contract but yielded passively to pressure. At this time, as has been already stated, the voice was still strong and speech unaffected in any way, but in the evening after a sleep of some duration this was no longer the case; it could scarcely be made out what he said. This was at first supposed to be due to dryness of the mouth or to the patient being only half awake, but it was soon seen to be an effect of paralysis.

Next day, April 10th, the affection of the speech was the first symptom to claim attention. It was such that without some clue to what the patient said it was impossible to understand him, but short answers to questions could be followed. There was no aphasia. He had words at command and could say them after a fashion; again there was no obvious paralysis of the lips or tongue. The lips moved in speech and he was able to drink, a little fluid escaping, but not more than might be expected in the helpless condition of the patient; the tongue was protruded at command and carried readily from side to side; the voice, moreover, was fairly loud and clear. The indistinctness of articulation was obviously due partly to want of breath, partly to want of power to maintain the vocal cords in apposition for phonation; a phrase was cut up into very short fragments each of which required a special effort, and only the beginning of individual words was actually pronounced.

The paralysis of the respiratory muscles had made rapid progress. When the trunk was exposed the alternating action of the chest and abdomen was now extremely conspicuous; as the upper part of the thorax rose the abdomen fell in, and as the thorax fell the abdomen rose, these movements being sudden and extensive; the diaphragm was simply flapping to and fro. The lower ribs also, which on the previous day had expanded as well as risen, were now drawn in during inspiration. Of course under these circumstances there was very little entry of air into the lower part of the lungs, and the respiratory murmur was scarcely audible.

There was no very marked aggravation of the paralysis of the limbs; both arms and legs could still be moved slightly, the legs better than the arms. Tickling of the soles excited movements and exclamations, and sensation was apparently unaffected.

The hands were scarcely as much swollen as on the previous day; the feet not at all. The extremities were warm.

The urine was passed naturally.

The expression of the face was natural and the colour good.

The pulse had the same character as before; short, weak, soft, and small. The first sound of the heart was short, sharp, and fairly loud, the second extremely feeble over both aorta and pulmonary artery.

Later in the day he slept, and as before was much worse on waking; the speech more indistinct and the paralysis of the respiratory muscles aggravated. Râles began to be heard in the bronchi and trachea. From this time there was a steady advance of the symptoms, and death took place about noon on April 11th.

Permission to examine the spinal cord was obtained, and this was done at 9 a.m. on the 12th, twenty-nine hours after death, before the body was cold.

The thin legs contrasted with the somewhat bulky body and well-developed arms, and the experienced assistant spontaneously called attention to the extraordinary whiteness and the swollen condition of the hands, and to the wet and sodden state of the structures of the back.

When the spinal canal was opened there was nothing noticeable outside the sheath of the cord, and the dura mater was quite normal. The cord itself, again, when exposed was firm to the touch, and the only abnormal appearance was its whiteness and the small size of the vessels on its surface.

The most careful microscopic examination of stained and hardened specimens failed to detect any structural change in the cells or fibres.

The first case of the kind which came under my observation was in St. Mary's Hospital while I was resident medical officer, but I have been unable to find my notes of it. The patient was a young woman who had taken to drinking after being deserted. Other cases more or less similar seen at different times may be briefly mentioned.

One was that of a young married lady seen with Dr. Myrtle, of Harrogate, in April, 1882, and described by him at the

meeting of the British Medical Association in August of that year. The lower extremities had been slowly losing power but could still support the weight of the body; the extensors of the wrist and hand were beginning to show weakness, but the most striking and characteristic feature was the complete loss of tone in the capillaries, so that when the feet were allowed to hang down they could be seen to fill with blood and become turgid and swollen.

A widow lady, æt. about 50, long addicted to alcohol and who for two or three years after the death of her husband had indulged in brandy unrestrainedly, was seen in consultation with the late Dr. Rhodes, of Weymouth. She was in bed, but still able to use her legs and support her weight upon them, while the hands had become useless, and, as she held them out, dropped at the wrist and could not be extended. They were pale and puffy, the dorsum being swollen and rounded as in dropsy. The muscles generally were extremely soft and tender. There was no disease of heart or kidneys. This patient died about a fortnight later from gradual paralysis of the respiratory muscles apparently beginning with the diaphragm.

A case seen with Mr. G. Amos Duke was at a more advanced stage. The patient, æt. 31, was the wife of an innkeeper who had for two or three years taken at least a bottle of brandy per day. A child to which she had given birth seven years before was said to have manifested syphilis, and she had had repeated miscarriages since.

For three months her legs had dragged, and three weeks before our consultation she was said to have had sudden loss of power in all her limbs, with speechlessness and difficulty of swallowing. She was, however, away from home at the time, and not under observation, and had travelled from Eastbourne to London afterwards, without difficulty. For a fortnight paralysis had gradually been coming on, and when seen on November 9th, 1878, there was marked loss of power in the upper extremities generally, with dropping of the wrists, great impairment of power also in the lower limbs, without

absolute paralysis, while the diaphragm was apparently completely paralyzed, respiration being thoracic only. There was aphonia, partly from want of breath, but also from some degree of laryngeal paralysis; liquids entered the larynx and provoked choking and cough. The movements of the soft palate were natural, and the tongue was well protruded. She died shortly afterwards from asphyxia.

In the case just spoken of there was a suspicion of syphilis, and another patient seen January 1st, 1884, with Mr. Wheeler, near Kew, was actually under treatment for this disease, which he had contracted in March or April, 1883. He was thirty-nine years of age, but looked forty-nine at least, and had been a heavy drinker from the age of twenty, taking at times extraordinary quantities of spirits. At the end of October, when he came under Mr. Wheeler's care, his legs were weak and shook under him as he walked. This had been noticed for a fortnight, and when he sat down he had needed helping up. About the middle of November he became unable to walk, and first the left and then the right hand became paralyzed, the paralysis beginning at the ulnar side. When I saw him on January 1st he lay helpless in bed; the legs could be feebly drawn up, but could not be put down again without assistance, the upper extremities were helpless but not immoveable, there was slight paralysis about the mouth, and the diaphragm was paralysed. Sharp pains shot down the legs from time to time, and there was incontinence of urine, these latter being exceptional features. The hands and feet had the pale puffy appearance mentioned in other cases, and the muscles were soft and flabby.

The paralysis invaded the walls of the chest, and the patient died on January 8th.

In trying to assign to this form of paralysis its proper place among the diseases of the nervous system, it will be well to recapitulate briefly its characters and peculiarities.

There is first gradually increasing weakness of the lower extremities which may be noted for some time, when marked loss of power becomes manifest in the extensor muscles of the forearm giving rise to double drop-wrist. It cannot be said that when this is first apparent there is no distinct paralysis elsewhere; the lower extremities and the back are weak, but the drop-wrist may be complete, and the flexors of the hand may also be paralysed so that the hand is like a flail, while the movements of the elbow and shoulder are good and fairly vigorous, and the patient can support himself on his feet. As the paralysis advances all the muscles of both upper and lower extremities are affected, and the limbs can only just be moved, the arms being as a rule more seriously implicated than the legs; the muscles of the trunk also are paralyzed so that the patient cannot raise himself or turn in bed. The sphincters retain their functional power, and sensation is not affected. There is no pain from first to last, though the muscles may be tender on handling. An exception to these statements, however, is furnished by the case last seen. No convulsion or jactitation occurs.

Death is caused eventually by paralysis of the respiratory muscles, and the diaphragm is the first of these to be affected. Instead of descending during inspiration it remains flaccid and is forced upwards by atmospheric pressure on the abdomen; in expiration the reverse of this takes place, so that the chest and abdomen no longer act together in respiration, rising and falling simultaneously, but their movements alternate. As the chest expands the abdomen falls in, and as the chest contracts the abdomen is protruded. The thoracic movements proper are the more energetic in order to compensate for the loss of the diaphragmatic respiration, so that the see-saw action of the chest and abdomen is very conspicuous, and at first there is actual expansion of the lower segment of the chest. Later the muscles of the chest become affected, apparently from below upwards, when, of course, death ensues from apnœa.

A striking feature of this form of paralysis is the loss of tone in the capillaries. It has been stated in the description of the case (p. 135) that when the patient was seated on the edge of the bed with the legs hanging down, the feet were filled with blood and could literally be seen to swell and become puffy. This was equally conspicuous in Dr. Myrtle's case, the lividity being more marked. The capillaries yield and permit themselves to be distended. The pale or livid and swollen condition of the hands, when they could no longer be raised but lay in the bed by the side of the patient, was due to the same cause, as was also the unusual degree of saturation of the structures of the back with moisture found after death. It is the degree of capillary paralysis which is remarkable, and the early period at which it is observed; almost all paralyzed limbs swell when allowed to remain long in a dependent position, but in no other form of paralysis is the stagnation of blood so complete and immediate or the swelling so rapid, and it is to be noted that this is observed when the loss of power is not very considerable.

There can be no question as to the seat of the disease giving rise to the symptoms described; this is obviously the spinal cord. As to its nature no discussion is required to exclude myelitis, diffuse or localised. Very brief consideration again is sufficient to show that the affection is not so-called acute anterior poliomyelitis occurring in adults; the regressive paralysis of Dr. Barlow, which is never fatal by paralysis of the respiratory muscles; it is strikingly progressive instead of being regressive. The disease which it most resembles is acute ascending paralysis, and it is probable that the condition of the cord is essentially similar, no lesions being discoverable by any mode of preparation at present known. The features common to the two are the progressive advance of the paralysis till death is brought about by paralysis of the respiratory muscles; the absence of fever or pain or spasm; the fact that sensation is not affected and that the sphincters retain their functional activity. The

principal difference is the order in which the muscles are invaded.

In acute ascending paralysis the legs are first affected and the loss of power gradually extends from below upwards; this becomes particularly noticeable when the chest is reached; the intercostals are first attacked and respiration is carried on by the diaphragm after the thorax has become motionless. The only case of acute ascending paralysis which I have seen presented a very different clinical picture when the stage of dyspnœa was reached to that observed in these cases. The patient could not lie down, but insisted on being supported in the sitting posture with the arms well raised by attendants so as to draw up the chest, while the head, which the neck was no longer able to support, had to be steadied and prevented from falling forwards or to one side; another difference appears to be that acute ascending paralysis is more common in men than in women. The cases collected by Erb[1] are twelve men and four women, while alcoholic spinal paralysis is more frequently seen in women. I find no note by observers of loss of tone in the capillaries in acute ascending paralysis, and I did not remark it in the case I first saw.

Admitting the similarity between the condition under consideration and acute ascending paralysis, I think the difference sufficiently marked and constant to warrant a distinction by means of a name. This name I should take from the etiology. In all the cases which have come under my observation there has been excessive indulgence in alcohol, and I have no hesitation in assigning this as the cause of the disease, the alcohol exerting an influence on the grey matter of the spinal cord probably similar to that which in the brain gives rise to delirium tremens.

A point worthy of note is that in all the cases except that which forms the subject of this paper and that seen since it was written the patients were women. Dr.

[1] 'Ziemssen's Handb. der speciellen Pathologie und Therapie,' Bd. xi, 2te Hälfte, 2te Abth., S. 324 (Translation, vol. xiii, p. 735).

Rhodes's and Dr. Myrtle's patients were ladies in good position; Mr. Duke's the wife of a publican; the hospital patient was the discarded mistress of a rich man. It is not probable, however, that sex is the factor which determines the direction taken by the injurious effects of alcohol. The affection of the spinal cord does not apparently occur among the women who are admitted into hospitals, or it would have been frequently observed and described; that is, it does not occur in women who have to work for their living or who have laborious domestic duties. It would appear that unrestricted access to stimulants throughout the entire day, and an indoor inactive mode of life, were the conditions which together invited the disease; these were just the conditions common to the five women and two men who have come under my observation.

NEURECTOMY OF THE SECOND DIVISION OF THE FIFTH NERVE.

BY

THOMAS F. CHAVASSE, M.D., C.M. (Edin.),
SURGEON TO THE BIRMINGHAM GENERAL HOSPITAL.

Received October 20th, 1883—Read February 26th, 1884.

Of the sensory nerves affected with neuralgia, the branches of the second division of the fifth are most frequently involved, for in their course through osseous canals they are prone to be subjected to pressure, whilst their peripheral terminations are exposed to varying atmospheric influences.

In the two cases, notes of which I bring before the Society to-night, the posterior dental branches of the superior maxillary nerve appeared to be the starting-point of the pain. Both patients in their younger days had been addicted to alcohol and both had been accustomed to work in heated atmospheres. There is no history of syphilis in either case.

J. L—, æt. 50, came under notice July 19th, 1882.

History.—Fourteen years ago, after a heavy drinking bout, suddenly felt severe pain in the upper molar teeth of the right side, this being accompanied by a series of fine colours (blue, red, green, &c.) visible only to the eye on the affected side. There were no decayed teeth, but as the pain persisted the last upper two molars, supposed to be the exciting factors, were removed; this procedure

afforded no relief. Paroxysms of pain have been of constant occurrence since the first onset; the pain is described as being of a jumping character of short duration. Of late years the attacks, when they occur, have lasted longer. Sometimes the spasms are felt only five or six times daily, at others they follow in quick succession for the entire twenty-four hours. Atmospheric influences more readily affect them than anything else, exaggeration taking place in cold wet weather. Talking and movements of the jaw during mastication will also act as irritant causes. The patient has endeavoured to follow his employment at intervals, but not very successfully; for the last six months he has been quite incapacitated, and owing to disturbed sleep and constant pain there has been a considerable loss of flesh. The man has been an inmate of various hospitals and has been dismissed as incurable. Many drugs have been tried, and with the exception of opium have failed to procure relief; the same may be said of blistering, cauterization, and galvanism.

I treated the man as an out-patient of the Birmingham General Hospital for six weeks, and in September, 1882, admitted him.

It was then seen that the description of the pain had not been exaggerated; that the first sensations of a painful paroxysm were referred to the periphery of the posterior dental branches of the second division of the right trigeminus, at the point where the molar teeth had been removed; the pain then seemed to pass up to the temporal fossa and was felt there and in the lower eyelid, by the side of the nose, in the cheek, and the upper lip. This course was nearly always exactly followed.

During an attack the right eye became injected and suffused, the tears streaming over the face. When it rained, or threatened to do so, the man used to roll upon the floor in his agony. Morphia subcutaneously only afforded relief for a very short time. The patient was a strongly built man with healthy viscera, and when free from pain had a good appetite.

October 3rd, 1882.—I performed the following operation: An incision was made from the inner to the outer canthus of the right eye along the floor of the orbit. Another longitudinal one was made from the centre of this through the substance of the cheek, curved at its lower extremity and terminating at the angle of the mouth on the right side. The flaps thus marked out were reflected, and a dissection made to expose the infra-orbital nerve as it emerged from its foramen. This having been laid bare, a half inch trephine was applied to the antrum and its anterior wall partly removed.[1] The posterior wall of the cavity was next partially removed by a quarter inch trephine and the spheno-maxillary fossa thus opened. At this period of the operation the hæmorrhage was profuse. The bony canal containing the infra-orbital nerve was next broken up by means of a small chisel and a pair of scissor-pliers. The nerve was then traced back with considerable difficulty to the spheno-maxillary fossa, the posterior dental branches divided, and Meckel's ganglion exposed. With a long curved pair of scissors the trunk of the second branch of the fifth nerve was divided in front of the foramen rotundum and taken away with the ganglion. An inch and three quarters of nerve was thus removed. The portion in front of Meckel's ganglion appeared quite healthy; that behind was red and injected, although the usual size.

The hæmorrhage at certain parts of the operation was severe, especially at the opening of the spheno-maxillary fossa. The wound was plugged with boracic lint, the end of the strip being brought out at the lower angle. The edges were then approximated by sutures.

There were no constitutional symptoms following the operation, and none of the old pain was experienced, although on the third day there was a heavy rainfall. By October 16th the wound had healed.

On testing sensation, patient states that the most

[1] After removing the anterior wall of the antrum a reflector and artificial light will greatly assist the operator.

numbed parts of the face are the right side of the upper lip, and the structures lying over the lower wall of the orbit on the affected side.

Along the side of the nose and on the cheek, external to the longitudinal incision, sensation is less marked than on the corresponding side, according to the idea of the patient as 1 to 2. On examination of the soft palate there was no evidence of sensation being more marked on one side of the uvula than the other. A month later the statement was made that the feelings of sensation in the face have been gradually reviving. At the present time, however, they do not equal those on the normal side. The patient has continued free from pain and follows his former occupation.

J. P—, æt. 46, warehouse packer, admitted to the General Hospital, December 27th, 1882.

History.—When young was a heavy drinker, and has worked much in hot rooms. Has had smallpox, typhoid fever, but denies syphilis.

Eleven years ago, while at work, experienced a severe jumping pain in the upper molar teeth of the left side. This was followed by a copious flow of tears from the eye on the same side. The painful spasm was subsequently repeated; at first it was momentary and only felt at intervals of some hours, but it soon occurred more frequently and would be repeated five or six times in an hour; the intensity of the pain was often so great that it would awaken from sleep.

It could be elicited by speaking, masticating, and walking rapidly, and always by washing the face with cold water. The removal of the upper molar teeth seemed to give relief for a short time, but the patient gradually lost flesh, appetite, and sleep, although vigorously treated by varying methods in various hospitals, by different private practitioners, and numerous quacks. The severity of the pain at last became so great that it compelled the man to lie continuously in bed.

On admission.—The patient was found to be pallid and emaciated, with an anxious careworn countenance. He was unable to eat solid food owing to the pain induced by movements of the jaws. To walk across the ward or to talk produced a paroxysm. Continuous pressure on the site of the upper left molar teeth would induce the same. The pain is referred to the left temporal fossa, to the eye. and the side of the nose. During an attack there was no manifest injection of the ocular vessels, and no epiphora.

February 9th, 1883.—An operation similar to that described in the last case was performed.

After laying bare the infra-orbital nerve, a spartan grass, such as is used for pipe-cleaning, was inserted into the infra-orbital canal to act as a guide. The anterior wall of the antrum was not very thick, but the cavity itself was abnormally deep. Profuse hæmorrhage followed removal of the posterior wall.

The posterior dental nerves were seen and divided, but owing to the bleeding and the depth of the antrum, Meckel's ganglion could not be distinctly defined. An inch and a half of the nerve trunk was exsected.

More constitutional disturbance followed the operation than in the preceding case, the patient experiencing a bad attack of nasal catarrh. By February 26th he was able to eat solid food without inconvenience, walked about, talked, slept, and felt no pain.

March 5th.—On testing sensation, it was found that on the affected side there was a diminution of sensibility along the side of the nose, the lower eyelid, and the left portion of the upper lip. In the soft palate no abnormal appearance can be detected.

The man now gains his own livelihood and continues well. Occasionally, he informs me, especially when the face is exposed to anything cold, a slight momentary twinge of pain is experienced, but this subsides immediately the irritant is removed.

July 1st, 1884.—Both patients remain well.

Carnochan, of New York, who was the first to advocate

this operation, thought it was best performed by making a V-shaped incision with the apex at the infra-orbital foramen. The nerve having been laid bare, a curved sharp-pointed bistoury was passed into the mouth and made to pierce the substance of the cheek at the apex of the V, and the parts were then divided to the angle of the mouth. As there seemed to be no great advantage obtained by opening the cavity of the mouth, while there were evidently a good many disadvantages, in my own cases the plan of procedure was so far modified as to leave the oral mucous membrane intact.

In Carnochan's article on "Exsection of the Second Branch of the Fifth Pair of Nerves"[1] the following sentence occurs:—"I believe that, in such aggravated cases of neuralgia, the key of the operation is the removal of the ganglion of Meckel or its insulation from the encephalon." This from a physiological point of view is a dubious deduction, for, according to Brown-Séquard, pinching of the ganglion in animals fails to produce any evident sensibility, and, according to Prevost, none at all, though tearing it from its normal situation gives rise to very acute pain, which, however, may be the result of laceration of surrounding sensory fibres.

Be this as it may, most surgeons who have followed Carnochan have destroyed Meckel's ganglion.

In a case operated upon by Billroth the body was not touched, but this surgeon says, "In similar cases I should consider the removal of this ganglion of importance."

In thinking over my first case the question of treatment by nerve-stretching, neurotomy, and neurectomy had to be decided. Nothing could be simpler than to stretch the infra-orbital nerve as it emerged from its canal on to the face, but Valentine and others have shown that the stretching of a nerve-trunk merely diminishes the reflex excitability of its peripheral termination, and the traction so exerted is not conveyed to the central origin of the nervous trunk. In both the cases here recorded, the commencement of the

[1] 'American Journal of Medical Sciences,' n. s., vol. xxxv (1858), p. 136.

pain was invariably referred to the periphery of the posterior dental branches, and it appeared very doubtful if the stretching would have any effect on slender branches at some distance from the extension point. By dissecting out the whole nerve it was reasonable to assume that the same result would certainly be effected as by the milder procedure, together with anything more favorable which complete removal might possibly afford.

With regard to von Langenbeck's plan of dividing the nerve in the spheno-maxillary fossa by passing a long tenotome beneath the external palpebral ligament and along the outer wall of the orbit, it seemed doubtful if the nerve-trunk could be divided for certain behind the posterior dental branches, and in fact the operation would be merely a cut in the dark. Besides, cicatricial contraction of the central stump might subsequently act as a renewing irritant, and removal of only a small piece of a nerve-trunk may finally end in a reproduction. I have had a case under my care in which the inferior dental nerve reunited after a quarter of an inch of its trunk had been exsected.

I have tabulated, excluding my own patients, 22 cases in which Carnochan's operation has been performed. The following is the result:

Temporary relief was obtained in all.

(4, 7, 12). In 3 relief appears to have been permanent.

(5, 6, 8, 15, 19, 20). In 6 relief appears to have been of long duration.

(9, 13, 16, 18). In 4 relief appears to have been of short duration.

(1, 2, 3, 10, 11, 14, 17). In 7 the ultimate result is doubtful.

In 2 sufficient time has not elapsed to enable a correct estimate to be formed.

The doubtful cases were lost sight of; in two of them no return of the pain had been noted in twenty and fourteen months; in two more for one year; in another complete relief as regards the pain proceeding from the

superior maxillary nerve. Two never returned for further treatment, no pain being felt after two months and one month respectively. There is no record of a fatal termination. I think with all fairness it may be assumed that some of the doubtful cases may be regarded as permanently relieved, for if there had been a relapse we should in all probability have heard of them again.

An operation for neurectomy of the superior maxillary nerve, not at present much known in this country, is that which has been devised by Professor Lücke, of Strasburg. An oval incision is made from a point just above the external canthus of the eye, passing at first backwards, then downwards and forwards, and terminating at the zygomatic process of the upper jaw. The masseter muscle is divided and the zygomatic arch sawn through anteriorly and fractured posteriorly. This piece of bone, with the temporal fascia attached to it, is turned upwards. By these means the spheno-maxillary fossa is reached and the nerve is cut as it emerges from the skull. The fractured bone is then replaced and the masseter muscle attached to it with sutures. Union of the bone shortly takes place. Professor Lücke informs me that he has performed this operation three times with satisfactory results. The drawback to it, however, is the contraction of the muscle which is apt to follow, leading to depressed cicatrices, and thus necessitating prolonged after-treatment.

To obviate this difficulty Professor Lossen, of Heidelberg, has modified the operation by dividing the temporal fascia along the upper edge of the zygoma, then, after fracturing the bone, turning it backwards with the masseter left intact. After replacement of the bone, the temporal fascia is stitched in its old position, and the masseter is unable to draw the bony fragment downwards. In ten or twelve days the patients are able to open the mouth slightly, and eventually the full movement of the inferior maxilla is restored; but little deformity results as linear cicatrices mark the lines of incision. Braun, in the 'Centralblatt für Chirurgie,' April 22, 1882, records five cases of

intractable neuralgia operated upon by this method with the following results:

Case 1.—Man, æt. 40. Pain of four years' duration. Remained well two and three quarter years after operation.

Case 2.—Man, æt. 62. Pain of eight years' duration. Remained quite well for nine months, then paroxysms returned, but were less frequent and less severe than before the operation.

Case 3.—Man, æt. 46. Duration of pain not stated. No return of pain for ten months. Then a paroxysm was induced by very cold weather, but soon disappeared and has not reappeared. (Date of operation, February 3, 1881.)

Case 4.—Woman, æt. 42. Pain of nine years' duration. No return in nine months.

Case 5.—Man, age not stated. Pain of twenty-two years' duration. No return in four months.

Reyher, of St. Petersburg, reports the case in the 'Centralblatt für Chirurgie,' Sept. 2nd, 1882, of a woman, æt. 72, very stout, with neuralgia of the right trigeminus commencing thirteen years before. Had had neurectomy performed by Wegner's method, and neurotomy according to von Langenbeck, the operations giving relief for three years and two years respectively.

Reyher operated by first tying the right common carotid artery and then cutting away the nerve, according to the plan of Lossen, as it emerged from the foramen rotundum, this being rendered easy by the absence of bleeding. Seven weeks afterwards she is reported to be free from pain and the zygoma to be consolidated.

Nussbaum and Billroth have also cut away portions of the superior maxillary nerve by means of Langenbeck's osteo-plastic resection of the upper jawbone, and, still more recently, Gerster ('New York Medical Journal,' January 12, 1884) has advocated a modification of this procedure, by sawing through the middle of the malar bone. He thinks that by so doing access to the more central portion of the nerve is best obtained; he has so operated on three occasions.

Tabulation of Cases.

Operator.	Sex and age.	Duration of disease.	Date of operation.	Duration of relief.	Remarks.	Reference.
1. Carnochan	Man, 69	5 years	Oct. 15, 1856	No return of pain in 14 months	Superior maxillary nerve trunk found to be vascular, engorged, and thickened. Pain commenced in infra-orbital nerve	American Journal of Med. Sciences, Jan., 1858.
2. Carnochan	Man, 54	29 years	Oct. 10, 1857	No return in 7 weeks	For last 9 years pain had been continuous. Commenced in infra-orbital nerve. This had been divided by Chelius and other surgeons	Ditto.
3. Carnochan	Woman, 55	6 years	Nov. 5, 1857	No return in 1 month	Pain commenced in infra-orbital nerve	Ditto.
4. Wood, J. R.	Man, 42	Several years	April 2, 1866	No return in 2 years	Patient was lost sight of	New York Med. Journal, June, 1879.
5. Wood, J. R.	Woman, age not stated	Not stated	Sept. 29, 1869	9 months	Pain returned in inferior dental nerve. A portion of this removed, and after many months no return of pain	Ditto.
6. Wood, J. R.	Man, age not stated	5 years	June 29, 1870	„	Pain returned in inferior dental nerve. A part of this was removed with relief. In 1875 man returned addicted to opium, stating pain had reappeared. Died in 1875.—P.M. showed foramen rotundum closed by bone. No central changes in nerve origin	Ditto.
7. Wood, J. R.	Man, 50	10 years	Oct. 26, 1873	3 years	Patient was lost sight of	Ditto.

8. Blackman	Woman, 35	14 years	Jan. 24, 1868	20 months	Portion of infra-orbital nerve and the whole of the inferior dental had been removed previously. After return the paroxysms were as severe as ever	American Journal Med. Sciences, July, 1869, and Oct., 1870.
9. Mussey	Man, 32	5 years	June 11, 1869	2 months	Pain returned in inferior dental nerve, part of which was removed, but in four months agony as great as ever	American Journal of Med. Sciences, Oct., 1869, and Oct., 1870
10. Carson (Middleton)	Not stated	Not stated	Not stated	1 year	—	Blackman, Amer. Journ. Med. Sci., Oct., 1870.
11. Schuppert (New Orleans)	Man, age not stated	,,	,,	Pain returned in period not stated	Finally the carotid artery was ligatured and the facial nerve resected at styloid foramen. After one year no return of pain	Conner, American Journ. Med. Sci., Oct., 1870.
12. Schuppert	,,	,,	,,	No return after several years	—	Ditto.
13. Foote (Cincinnati)	Not stated	,,	,,	Pain returned in 3 months. Moderately severe	—	Ditto.
14. Fowler, S. R.	Man, age not stated	18 months	,,	Complete success as regards infra-orbital neuralgia	Reported that some pain is at times felt along the line of lower jaw and teeth	Proceedings Med. Society, King's County, 1877.
15. Cheever, D. W.	Woman, age not stated	Not stated	,,	2 years	Pain returned as badly as ever in the cheek, temple, and lower jaw. Infra-orbital region free	Boston City Hosp. Reports, Second Series, 1877.

Operator.	Sex and age.	Duration of disease.	Date of operation.	Duration of relief.	Remarks.	Reference.
16. Thorndike	Man, age not stated	Not stated	Not stated	1 month	Pain limited to infra-orbital region	Ditto.
17. Wagner, A.	Woman, 35	14 months	„	No return in 20 months	—	Langenbeck's Archives, vol. xi.
18. Wagner, A.	Woman, 45	23 months	„	Pain returned in 3 months	Neurectomy of infra-orbital previously performed. Central cause suspected	Schmidt's Jahrbücher, Band 146.
19. Weir, R. F.	Man, 47	24 years	Jan. 23, 1881	Remains free	Wagner's operation had been previously performed.	New York Med. Journal, Jan. 12, 1884, and private letter.
20. Markoe	Man, 30	Not stated	Dec., 1866	3 years	Neurectomy of inferior dental previously. In three years supra-orbital neuralgia appeared, but not severe	Ditto.
21. Howe	Woman, 45	12 years	May 29, 1883	Remains comparatively free	—	Ditto.
22. Gerster	Man, 36	18 years	June 16, 1883	„	Osteoplastic resection of upper jaw, and 1¼ inches of superior maxillary nerve removed two years before	Ditto.
23. Chavasse	Man, 50	14 years	Oct. 3, 1882	Remains free	—	—
24. Chavasse	Man, 46	11 years	Feb. 9, 1883	„	—	—

ON THE VALUE

OF

SYSTEMATIC EXAMINATION OF STILL-BORN CHILDREN.

BY

JOHN BLAND SUTTON,

LECTURER ON COMPARATIVE ANATOMY AND SENIOR DEMONSTRATOR OF ANATOMY, MIDDLESEX HOSPITAL MEDICAL COLLEGE.

Received February 22nd—Read March 11th, 1884.

For some considerable time it has been my custom to obtain, for purposes of anatomical and pathological inquiries, all stillborn children coming to hand in connection with the maternity department of the hospital. The object of this paper is to show that much valuable information may be gained by the *systematic* examination of fœtuses, as illustrated by a few selected cases possessing more than an ordinary interest.

In Dr. Peacock's well-known work on 'Malformations, &c., of the Human Heart,' 1858, at p. 114, the following sentence occurs:

"There is also conclusive proof, in some cases, that the valvular disease is congenital; for it has been found in infants which have died very shortly after birth and even in fœtuses which have never breathed; in other instances its intra-uterine origin may be fairly inferred from the precise

similarity of the diseased changes to those which are clearly congenital."

Further on he writes: "Analogy would lead us to expect what clinical experience demonstrates,—that the fœtus *in utero* is liable to diseases precisely similar in their nature and results, to those which affect the child after birth, and there can be no doubt that both the peri- and endo-cardium are not unfrequently the seat of inflammation during fœtal life."

These paragraphs, selected from Dr. Peacock's work, are really reflexes from the writings of other eminent pathologists who have "gone before;" they are given here as an embodiment of general opinion, but they require to be supported by actual facts.

In January last I received the body of a male fœtus born at the eighth month of intra-uterine life. It was well developed, but the skin presented the familiar gloss so characteristic of extreme anasarca. The belly was extremely distended. Not only was the subcutaneous tissue of the limbs, trunk, and scalp filled with fluid, but the peritoneal, pleural, and pericardial cavities were distended to the utmost by pale straw-coloured fluid.

All the viscera were duly and carefully looked to, but the only morbid conditions detected were large, soft, easily detached vegetations on the cusps of the valves guarding the aortic and pulmonary orifices, and a puckered condition of one of the curtains which form the mitral valve (Plate 1, figs. 1 and 2).

Here was a case of recent endocarditis, but from what cause arising? The mother had passed through several pregnancies in a satisfactory manner, had never suffered from rheumatic fever or any allied disease, and, as ascertained by two competent observers, the mother's heart was to all outward appearance and auscultatory tests perfectly normal. The husband and remaining children were in a healthy condition.

That several diseases may be transmitted from mother to child is beyond all disputation, *e.g.* syphilis; and the

celebrated case recorded by Hunter, in the 'Philosophical Transactions,' for 1780, of a "Woman who had Smallpox during her Pregnancy, and who seems to have Communicated the same Disease to the Fœtus," is a case in point. Dr. Edward Jenner has recorded similar cases in Vol. i of the Transactions of this Society.

In the fœtus, whose case I have recorded, no disease of the mother can be ascertained likely to cause endocarditis in her offspring, hence we are driven to the conclusion that *a fœtus may contract a disease independently of its mother!* In Dr. Jenner's case just mentioned it seems probable that one of the fœtuses contracted smallpox, whereas the mother escaped.

The case now to be related differs in its nature from the one just considered. It concerns the body of a female child which survived its birth three days, having been born at full time. Death was certified as being occasioned by "pneumonia."

On opening the abdomen the large size of the stomach attracted attention, and careful dissection disclosed the following state of affairs:

The stomach was three times larger than usual and presented a constriction at the ordinary situation of the pylorus. An inch further on, the intestine which was very dilated, suddenly terminated in a cul-de-sac having no communication with the surrounding viscera, even by fibrous tissue (Woodcut, fig. 1, p. 166). Immediately below this, and in contact with the head of the pancreas, was the upper extremity of the small intestine with the common bile-duct entering it.

Thus the continuity of the alimentary canal had been broken at the midlle of the duodenum, directly above the entrance of the common bile-duct. Below this spot the intestines were normal and as usual contained meconium, but this was small in quantity. The stomach held two ounces of fluid, in which brandy could be detected. On inverting the stomach the line of muscular fibre forming the pylorus was easily distinguished (see P, in Fig. 2, p.

166), and as represented in the figures is intended to give visual expression to this abnormal condition of things.

I am unable to advance any satisfactory explanation of this malformation, either on morphological grounds, or from anything known concerning the embryological history of the alimentary canal. It may bear some relation to the development of the liver, which commences as a diverticulum of the gut, for Dr. Wilks[1] and Dr. George Buchanan[2] have each recorded cases of partial obstruction of the intestine by a diaphragm at this point of the duodenum, above the papilla, which marks the entrance of the united ducts of the liver and pancreas. Mr. George Eastes[3] has also described a similar case in which the obstruction was complete.

Dr. Buchanan thinks it possible that the diaphragm-like structure encountered in his case resulted from the hypertrophy of one of the valvulæ conniventes. Mr. Pollock, in writing on congenital strictures of the small intestine, takes the same view, for in 'Holmes' System of Surgery,' 3rd edit., vol. ii, p. 712, in a foot-note we read:

"From the plate which accompanies the description given by Dr. Wilks, it would appear that the contraction in all probability arose from a highly developed valve of the intestine; in the centre of this valve or stricture may be seen a very small circular hole, apparently not larger than would admit a probe."

My object in reporting this case and making these references is to show that this view is absolutely untenable. After very many observations particularly directed to this question and to the date of the appearance of the valvulæ conniventes, it may be clearly demonstrated that *these folds of the mucous membrane do not arise until two or three weeks before birth.* I have *never* seen them before birth, but Mr. Shattock has found them quite rudimentary in the lower part of the duodenum and upper part of jejunum, in a well-developed fœtus at full term. If these observations be confirmed by independent workers, then

[1] 'Trans. Pathol. Soc.,' xii, p. 101. [2] Ib., xii, p. 121. [3] Ib., xxxi, p. 114.

the hypothesis, which would regard these abnormalities as examples of hypertrophied valvulæ, *no longer holds its ground.*

The third fœtus presents features of interest to those concerned with surgical pathology.

The drawings represent the spinal column, thorax, and rudiments of the upper limbs of a full term female fœtus (Plate I, fig. 5; Plate II, figs. 1 and 2).

The noteworthy points in the external conformation of the body are represented in Plate I, fig. 5. The upper limbs merely consist of a finger attached to each shoulder. On dissection, the clavicle is normal, the scapula well-shapen and of normal size, but in place of a glenoid fossa it presents a rounded head of hyaline cartilage. To this succeeds a cartilaginous representative of the semilunar bone, followed by an os magnum also in cartilage; articulating with this is a metacarpal bone, supporting in its turn three phalanges. Humerus, radius, and ulna are not represented.

The spinal column presents a considerable curvature, involving the whole column, the concavity of the curve looking to the left. The unnatural bend commences about the third lumbar, and extends, increasing in severity, to the mid dorsal region; it then ascends to the neck region, ending about the second or third cervical vertebra. The bones composing the column are normal in number, but the centra are narrower on the concave than on the convex side of the curve. The thorax has the usual deformation seen in lateral curvature; for example, the chest is deeper and the intercostal spaces are wider on the convex than on the concave side of the curve, exactly as one sees it in instances of the disease occurring in children.

There are several cases on record of congenital lateral curvature, but in all these cases the curves result from the interpolation of segments or half vertebræ; thus in a case Rokitansky records in his 'Pathological Anatomy,' vol. iii, p. 230, there were four half vertebræ, with their

half arches and processes, too many. They were so placed on the two sides as to compensate one another, and cause four curves in the spine; two in the dorsal, one in the dorso-lumbar region, and the fourth involved the sacrum. But in the case described by me no such condition can be detected.

In connection with this case it will be well to mention that the Middlesex Hospital Museum contains a well-prepared skeleton of a presumably full-grown female, in which there is a lateral curvature of the spine involving the lumbar region with a second curve in the dorsal portion of the column. In this skeleton eleven vertebral bodies exist in the dorsal series, but there is the normal number of spinous processes, so that the curve in the dorsal region results from the deficiency of a portion of a vertebral centrum, and as there is no evidence to lead one to suppose the defect to be due to disease, it must, therefore, be regarded as a congenital defect. This is not all, for in this self-same skeleton the right thumb bones are exceedingly small and dwarfed with defect in the number of the carpal bones, and on the left side the radius is absent and so is the thumb, whilst the ulna exists as a short and exceedingly curved bone.

One question intrudes itself on the mind engaged in meditation on this adult skeleton and the fœtus represented on Plate II, fig. 1.

Does any relation exist between congenital curvature of the spine and defective development of the upper limb?

This question suggests another, Are the two conditions so frequently associated as spina bifida and club-foot?

The query cannot be answered until more observations have been recorded.

The remaining cases of interest will be treated briefly. Three fœtuses presented abnormal conditions of the kidneys.

The first was an example of single kidney, the left one being completely wanting, no trace of ureter, or anything

to represent the renal artery or vein of that side. The right kidney was twice the usual size, and the ureter was very large and dilated. The urethra and bladder were normal.

The remaining two were examples of cystic kidney, not hydronephrotic, due to interference with the outflow of urine from ureter or urethra; but the kidneys were enlarged and made up of a congeries of small cysts, the largest of about the size of a pea. The ureters, bladder, and urethra were normal. In one of these specimens the kidney was of the "horseshoe" variety. In the other specimen, the kidneys were of the usual shape, but presented an interesting condition in relation to the left testis. The occurrence of intra-uterine peritonitis has long been recognised as one cause of retained testicle; in this specimen the right testicle was in its normal situation, but the left one was firmly attached by fibrous tissue to the lower end of the corresponding kidney, the inguinal canal was patent, and the funicular pouch of peritoneum occupied the scrotum. The matting together of coils of the intestines and the thickened condition of the peritoneum spoke plainly of peritonitis.

Deficiency of the diaphragm.—When we reflect that one of the chief characteristics of mammalian myology is the diaphragm, anything concerning its defective development must be of interest. Many cases of partial deficiency of this muscular septum are recorded; to these I add another. The fœtus was born at full term, and on examining it I found that the left lobe of the liver filled up the corresponding side of the thorax, it having passed through a hole caused by the defective development of the diaphragm. The only representative of the left lung was a small piece of pulmonary tissue an inch square. The remaining viscera were normal.

Cleft palate was seen in three instances, one of which was associated with a rare defect in the sphenoid bone, viz. absence of the floor of the pituitary fossa. This is important in its bearing on the occasional occurrence of

meningocele, bulging into the upper part of the pharynx and nasal fossa. That such a defect should occur in this situation may be readily comprehended, seeing that the fossa in question marks the spot where the trabeculæ cranii separate to enclose the pituitary body.

Meningo-encephalocele occurring in the occipital region was seen once, and this leads me to offer a few remarks on a neglected point in the anatomy of this part of the skull which has some bearing on meningocele in this situation.

If the skull of a human fœtus be examined about the third month of intra-uterine life, the foramen magnum will be found formed in the following manner: In front is the cartilaginous basi-occipital with its nucleus just appearing; on either side are the ex-occipitals, with their centres visible in the mass of cartilages forming the greater portion of the side-walls of the skull; and posterior to these is a membranous space. These relations of parts may be seen on reference to Plate I, fig. 4. This space or fontanelle extends upwards to the situation of the occipital protuberance, and during early development serves to separate the two inferior centres of this portion of the bone, for the squamo-occipital arises from four centres, the two upper ones originating in membrane, and the two lower ones in cartilage, the four pieces eventually becoming confluent at the situation of the protuberance. This membranous space, which I propose to term the "temporary occipital fontanelle," is really a remnant of the original membranous roof of the cranium. During the fourth month, the two inferior centres of the squamo-occipital fuse together and send down a bony spiculum to occupy the fontanelle, as see in Plate I, fig 3. Kölliker figures this spicule of bone in his 'Entwicklungsgeschichte,' 1876—79, p. 450, fig. 278, but the membranous space has evidently escaped his observation. By the commencement of the fifth month, the fontanelle has become filled and the foramen magnum is bounded as usual by the squamo-occipital. From these statements it is obvious that from

the beginning of the third to the end of the fourth month of intra-uterine life, there exists a fontanelle extending from the foramen magnum, and when at its greatest relative size reaching into the occipital protuberance. The practical bearing of the case is this: *in all probability it is this membranous space or temporary weak spot in the cranium that explains the frequent occurrence of meningocele and encephalocele in this situation.*

It would prolong this paper indefinitely to describe all the cases of interest which have come under my notice during the past two years. I have contented myself by giving the details of those which seemed, on account of their rarity and scientific bearing, to be of interest to the physician and surgeon.

Concerning the diseases of the fœtus we know but little. Malformations of the heart and fœtal abnormalities in general have received a "lion's share" of attention. I feel that much good light may be shed concerning hereditary diseases if the bodies of stillborn children be as *systematically* examined in our hospitals, as are the patients who pay the "debt of nature" in these institutions.

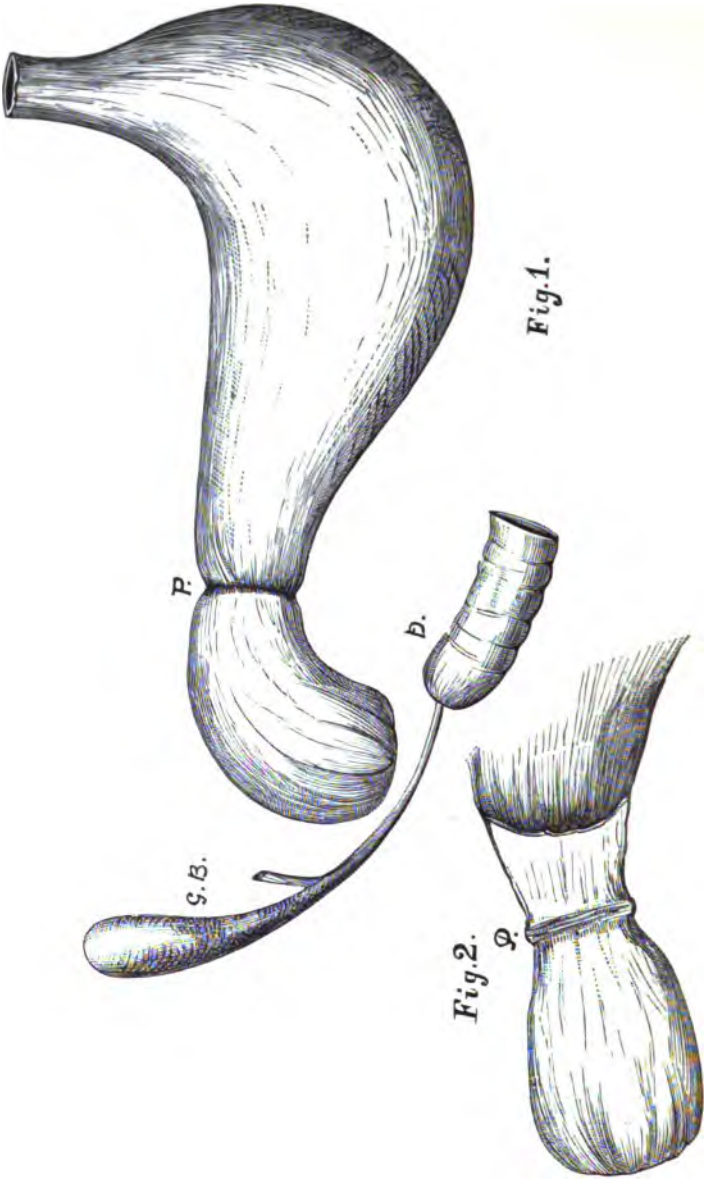

P.
b.
G.B.
Fig.1.
Fig.2.
g.

FIG. 1.—Complete stricture of the duodenum immediately above the papilla for the united bile and pancreatic duct. P. Pylorus. G.B. Gall bladder. D. Duodenum.

FIG. 2.—The *cul-de-sac* inverted, showing a band of muscular fibres, (F) belonging to the sphincter. (Size of nature.) From drawings by Mr. Osmond.

DESCRIPTION OF PLATES I AND II.

On the Value of Systematic Examination of Stillborn Children. (JOHN BLAND SUTTON.)

PLATE I.

FIG. 1.—The pulmonary orifice of the heart of a fœtus, showing recent vegetations adherent to the valves. ($\frac{2}{3}$ the size of nature.)

FIG. 2.—The left ventricle of the same heart opened, to show recent vegetations on the cusps of the aortic valve and the puckered condition of one curtain of the mitral valve. ($\times \frac{2}{3}$.)

FIG. 3.—Posterior view of the skull of a human fœtus at the end of the fourth month. It is intended to show the "spicule" of bone extending into the membrane of the "temporary fontanelle." Other letters as before. S. The spiculum. (Twice the size of nature.) Semi-diagrammatic.

FIG. 4.—Posterior view of the skull of a human fœtus at the third month, to show the temporary occipital fontanelle, F. S.O. Squamo-occipital. M. Foramen magnum. E.O. Ex-occipital. B. Basi-occipital. C. Chondro-cranium.

FIG. 5.—The external configuration of the fœtus, of which Figures 1 and 2 on Plate II represent the skeleton.

From drawings by Mr. Wynter.

PLATE II.

FIG. 1.—A drawing to illustrate a case of ateral curvature of the spine in a human fœtus.

FIG. 2.—The skeleton of the dwarfed limb from the same case. S. Semilunar. M. Magnum. M.C. Metacarpal bone. 1, 2, 3. The phalanges. (Natural size.)

From drawings by Mr. Osmond.

Fig. 1.

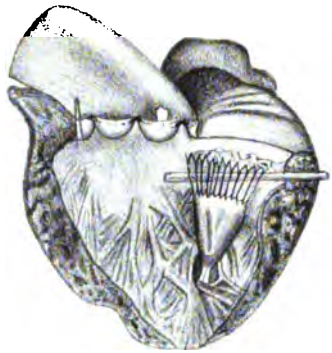


Fig. 2.

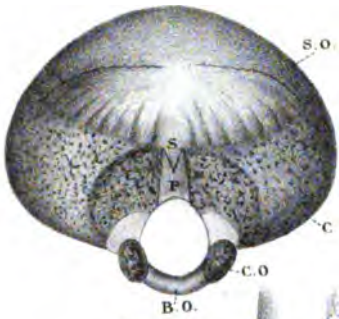


Fig. 3.

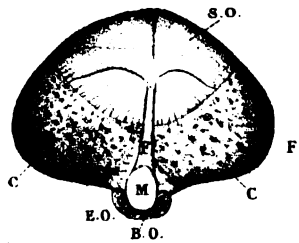


Fig. 4.

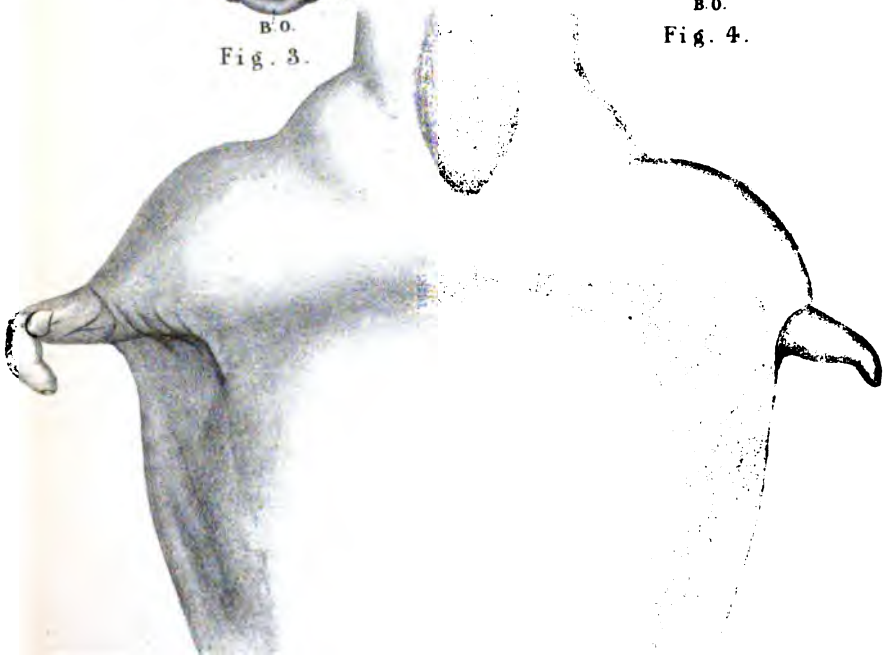


Fig. 5.

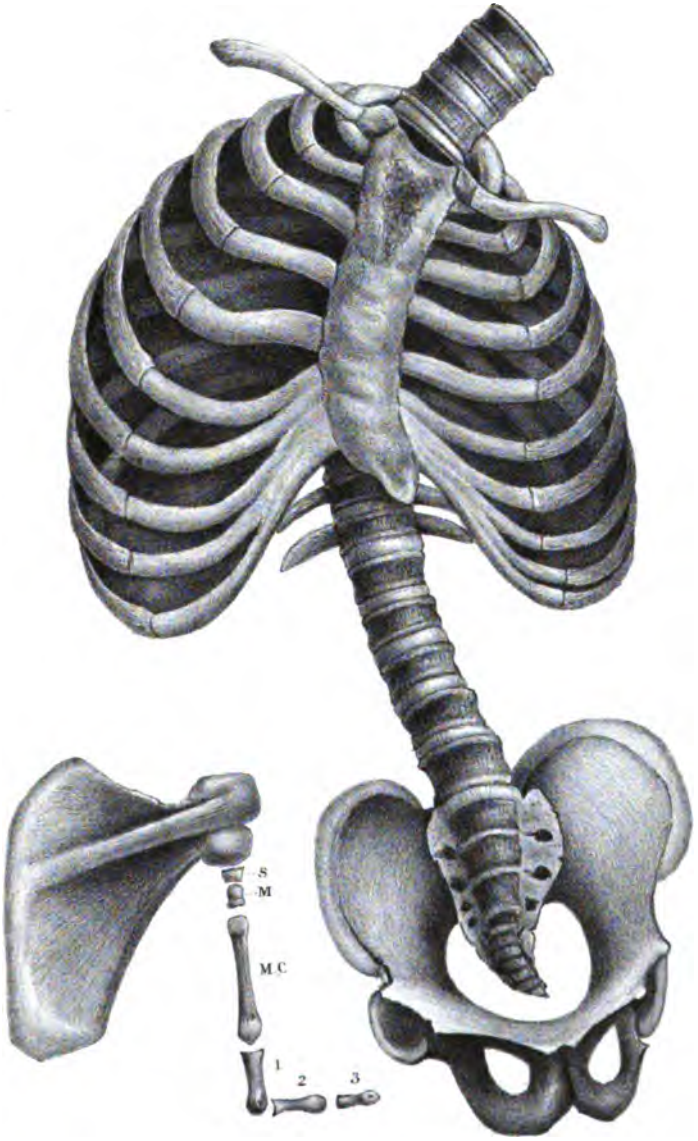


Fig. 2. Fig. 1.

Mintern Bros. lith.

THREE CASES

OF

XERODERMA PIGMENTOSUM, KAPOSI

OR

ATROPHODERMA PIGMENTOSUM.

BY

H. RADCLIFFE CROCKER, M.D., M.R.C.P.,

PHYSICIAN TO THE SKIN DEPARTMENT OF UNIVERSITY COLLEGE HOSPITAL; PHYSICIAN TO THE EAST LONDON HOSPITAL FOR CHILDREN.

Received January 8th, 1884.—Read March 25th, 1884.

THE disease of which I show examples to-night was first described by Kaposi in 1870, from four cases in Hebra's practice. Since then cases have been recorded in Germany, France, and America, but those now shown are the only cases known to have occurred in England, and were exhibited about a year ago at one of the Societies as examples of lupus. They, therefore, did not attract the attention they deserved and no account of them was published. I am indebted to Mr. Martin, a student of University College Hospital, for having them sent up to me.

The children are the three eldest of a family of four; the father, æt. 42, is a Shropshire farmer, and has always had good health, the mother, æt. 40, was in good health at the time of their birth and suckled them all. The last child, a girl, æt. 6 years, is perfectly healthy, and is described as having a "beautiful skin." This child

was not suckled. There is no history of cancer or any other hereditary disease in the family on either side.

Case 1.—Alice Elizabeth B—, æt. 12, was admitted with her brother and sister into University College Hospital on December 10th, 1883.[1]

She was quite healthy until she was between twelve and eighteen months old, when the disease commenced without any previous symptoms, with freckle-like pigment spots which appeared simultaneously upon the face, neck, back of forearms, hands, upper arms, and legs below the knees over the same areas that are now affected, and though the spots have increased in number and other lesions have appeared, the father does not think the disease has spread at all from the commencement. The lesions are thus limited to the uncovered areas, the child having worn short sleeves and socks, and the father thinks the disease began in spring or summer because the spots were thought to be sun-freckles.

Except that the lentiginous spots became more numerous, larger, and some deeper in colour, there was no alteration noticed until she was six years old, when ulcers appeared on the right cheek and nose.

Present Condition.—The patient is moderately well-nourished, but less so than her younger sister, does not suffer much pain, and her general health is fairly good.

Positions of Lesions.—The disease involves the whole of the face, ears, and neck, terminating on the forehead where the hair begins, in front, but in the temporal region the lesions extend in a minor degree back into the hair as far as a line drawn from the insertion of the ear. The scalp is, however, in great part covered with dirty brown scales of small size.

Below it extends to the level of the second rib in front and to the outer third of the clavicle at the sides and behind to the level of the fourth dorsal vertebra; it does not terminate abruptly, the pigments spots becoming gra-

[1] See Plate III.

dually more sparse below the level of the clavicles. It occupies the whole extensor surfaces of the fingers, hands, forearms, and arms as high as the insertion of the deltoid, but the nails are unaffected. On the flexor aspect the whole palmar surface is quite free, but that of the forearms is thickly covered, but more on the radial than the ulnar side, while it terminates abruptly at the wrist.

The inner aspect of the upper arms is nearly free, the affected areas being mapped out by a line drawn from the lowest point of insertion of the deltoid to each condyle of the humerus. On the legs the disease extends from the tubercle of the tibia, to three inches above the ankle in front, and for a corresponding area on the calf, but laterally, it goes up to the level of the upper border of the patella on the outer side.

The affected areas are quite symmetrical and the skin of all parts of the body not mentioned, is perfectly normal. The red part of the lips and adjacent mucous membrane is white, mottled with red streaks, but the tongue and rest of the oral mucous membrane are free. The right eye has a vascular pterygium extending from the inner angle to the cornea. The disease varies in intensity, being most marked on the face, neck, and extensor surfaces of forearms, while the legs are only slightly affected.

The lesions are multiform, consisting of:

1. Pigmented spots from a pin's head to an inch in diameter, many of the smaller being rounded and like ordinary freckles, but the larger are irregular from aggregation. They vary in colour from a light raw umber to a deep sepia, the large spots being the darkest. They are most abundant on the forearms and neck. Scattered amongst these lentigines are white atrophic spots about an eighth of an inch in diameter, in many parts scarcely noticeable, but in the upper part of the cheeks and in the infraorbital regions aggregated into larger areas of very white skin, very smooth and presenting a cicatricial aspect, or covered with thin layers of readily peeling epithelium. That there is actual contraction of the skin is shown by

the presence of some degree of ectropion of the lower lid, which is denuded of lashes; the mouth also is habitually open, though the lips can be closed. The surface of the skin is very finely wrinkled, especially round the mouth. On pinching up a fold it feels very thin and it is less easily effected than normally, but there is no approach to the difficulty experienced in a case of atrophic scleroderma.

There is marked cicatricial contraction of the nose, the nostrils being rounded and widely dilated, this contraction, however, being partly due to the ulcers, to be presently alluded to, and not only to the atrophy. Telangiectases exist as spots slightly raised above the surface from a pin's point to a third of an inch in size, of a bright crimson colour, and with a lens they can be seen to consist of small dilated vessels; they are not numerous, but conspicuous by their bright colour in the white atrophied skin. More abundant but less noticeable are fainter tufts of dilated capillaries.

Superficial ulcers covered with thick yellowish crusts are abundant upon the nose, and there are a few on the right cheek, but only two on the left. There is one as large as a shilling on the lower lip. When the crusts are removed the surface of the sore is slightly above the surrounding skin in some of them, below it in others. Three tumours are present on the right side of the face. The largest began a year ago on the tragus as "a little black lump;" it grew slowly, preserving its colour, and, when it was about the size of the end of the finger, began to ulcerate, six months from the time of its being first noticed. It is now a flattened spheroidal fungating mass four inches in diameter, projecting an inch and a half above the surface. It covers almost the whole of the right ear and is constantly exuding a sanious fluid. The second tumour, an inch and a half long and one inch wide, extends diagonally from the right malar eminence to the upper lip; it is evidently made up of two tumours fused together. The third is one inch in diameter, on the right side of the chin. These smaller

tumours are covered with black crusts of dried exudation. The tumours are tender, but are not often spontaneously painful. There are no granular enlargements.

More easily felt than seen are numerous small warts springing from the pigment spots; they are most abundant about the face and arms, and some of them closely resemble small pigmented senile warts. Insignificant as these warts appear they are of no slight importance in relation to the tumours.

All the viscera seem healthy, and the functions duly performed. The urine has a sp. gr. 1020, is acid, clear, with no albumen or sugar. Since the case was shown to the Society in January, my colleague, Mr. Marcus Beck, has been kind enough to remove the tumours, together with two glands beneath the large tumour, and a small piece of skin from the outer side of the upper arm. The indolent ulcers also were scraped with a sharp spoon. The large tumour, which was extremely friable and had grown considerably since it was shown to the Society, sprang from a comparatively narrow pedicle, about one inch in diameter, in front of the ear, and did not implicate any of the skin beyond the pedicle, which appeared to spring from the subcutaneous tissue.

After hardening in a fluid consisting of equal parts of a one sixth solution of chromic acid and alcohol, sections were made. The large tumour consisted of masses of papillomatous structure made up of elongated cylinders bordered with oval or cylindrical epithelium, enclosing smaller epithelial cells, with very little fibrous stroma (Pl. V). This structure was imbedded in granulation tissue which constituted the greater part of the tumour; some of the cells of this fibro-cellular tissue were spindle-shaped, and the whole tumour was tunnelled with numerous large blood-vessels. There was nothing approaching to a cancerous structure. The glands beneath were slightly enlarged, showing a slight increase of fibrous tissue; in short, only the appearances one would expect in a somewhat irritated gland. The smaller tumour showed more

epithelial structure. It, too, consisted largely of granulation tissue rich in blood-vessels, but the papilloma masses, instead of being congeries of separate cylinders, form digitate processes from a broader base; fibrous septa passed up between the lobes, so that it presented a gland-like appearance not unlike a molluscum contagiosum tumour in shape. The extremities of the processes lay free in a cavity in the granulation tissue, and in some places the masses had fallen out, leaving a lacuna in the granulation tissue.

How these gland-like structures are formed can be inferred from the small ulcer in which some of the epithelial surface was left.

In this there were only traces of the horny layer. The rete was increased in thickness, while the inter-papillary processes were enormously elongated by down-growth, and in some places they met and enclosed portions of the papillary layer of the corium. These hypertrophied inter-papillary processes apparently get separated from the rest of the rete, the compressed corium between the lobes forms the fibrous septa, and thus form the probable source of the pseudo-glands seen in the smaller tumour, and by still further disconnection with each other produce the papillomatous structure of the large tumour.

The rete-cells in many instances were vacuolated; there was leucocytic infiltration between the rete processes and through the corium and granulation tissue beyond it.

The skin showed atrophy of the papillary layer, the rete forming a slightly wavy line over the thinned corium. The horny layer was apparently unaltered, but the lowest layer of the rete was notably pigmented in parts with dark brown granules. At the site of a commencing wart, which was included in the section, the horny layers were much increased in number and separated from each other, and dipped downwards into a corresponding depression of the rete where the cells were flattened. In the centre of the wart the rete sent down an obtusely conical process into the corium; round this process was a scanty infiltra-

tion of leucocytes apparently proceeding from the vessels connecting the superficial and deep plexuses. There was no other alteration in the corium below the papillary layer.[1]

Case 2.—Alice Amelia B—, æt. 10, closely resembles her sister, but the disease is less advanced. Her nutrition is not at all affected. The disease began at the same age as Elizabeth's, viz. from twelve to eighteen months, and exactly in the same way and in the same situations, and has followed the same course with one exception. The sores began when she was only about four years old, within a month of their appearance in her elder sister.

The limits of the disease are the same as those of her sister except that in front it reaches only to the level of the first rib and behind to the second dorsal spine. On the forearms it is less marked on the ulnar side of the flexor aspect, does not extend beyond the first proximal phalanges of the fingers, and is much fainter upon the legs. There are two freckles on the right palm. The left leg has extensive superficial scars from a scald in infancy.

The pigment spots are about the same as in her sister, but the atrophic and vascular spots are not so numerous, and there are not such large white areas as in Case 1. The ulcers number about half a dozen on the face, the largest about half an inch across near the outer canthus, and they are nearly all on the right side. There is less cicatricial contraction than in her sister, the lips being perfectly mobile, but there is some left ectropion. The lids are granular, there is conjunctivitis, and great photophobia, but no pterygium. There are some verrucose projections to be felt both on the face and forearms, but no tumours at present.

Case 3.—Benjamin B—, æt. 9, is most like Case 2, but in some respects more advanced (Pl. VI). Beginning at the same age it followed the same course, but the ulcers

[1] The microscopical observations were confirmed by Mr. Marcus Beck, who concurred in the view than the tumours were papillomatous, and not epitheliomatous.

began when he was only three years old; in short, the ulcers began on all three children within a month of each other. The difference in distribution is, that it extends to the level of the clavicle in front and to the second dorsal vertebra behind, that the ulnar side of the flexor aspect of the forearm is almost free, and that the lesions extend to the end of the fingers. There are pigment spots on two of the nails of the right hand; the tip of the tongue has white spots upon it, where the papillæ are atrophied, so that it has a smooth pale aspect like some syphilitic tongues at that part. There are white spots also upon the lips. The pigment spots are like the others, but there are none more than three quarters of an inch across. There is a white smooth area over the right orbit and side of the nose, but the atrophic spots are inconspicuously scattered about amongst the pigment. Telangiectases are fewer than in the other; more than a dozen scabbed sores exist on the left side of the face, below the forehead, and on the nose, the latter being much distorted from cicatricial contraction. There are only a few small ulcers on the right side of the face. There are no verrucæ on the face, but a few on the arms. There are no tumours now, but there was one on the left cheek which grew out to the extent of an inch and a half in a finger-like way; it did not discharge, but the end became dead and was cut off from time to time, by the patient's mother. The stump was red but not ulcerating, and ultimately the tumour dropped off, leaving a slight scar.

There is a pterygium on the outer canthus of the left eye and on the inner of the right. The boy's general health and nutrition appear good.

Adding these to the already published cases, we have in all thirty-four cases on which we may found a general account of the disease. Taking first the etiological factors, we find that the number of males and females is exactly equal; consanguinity, however, plays an important part. The thirty-four cases are distributed over seventeen families, no less than twenty-six cases having occurred in nine

families. Another striking feature is its tendency to attack one sex only in a family.

This is true in seven instances. In Rüder's series, out of a family of eight boys and five girls, seven boys were attacked, while all the girls and one boy remained free. Nothing in the record of the cases suggests any explanation of this selection, and I do not remember any analogous instance where a disease, which is equally liable to attack both boys and girls, yet habitually selects only one sex in a family. Kaposi and myself alone record exceptions to this.

Age.—Nearly all the cases have commenced in the first or second year of life; one was only five months old. There are, however, two cases which did not begin until the age of nine and sixteen years respectively. The disease is obviously due primarily to a congenital predisposition, though, like ichthyosis and prurigo, it does not manifest itself for some time after birth, requiring perhaps some further condition for its development.

Heredity does not appear to have any influence. Three instances of cancer in seventeen families are recorded, but I should hesitate to regard that as of etiological significance.

Hygiene does not seem to have any influence. Most of the patients were in fairly good circumstances, and in no instance have the general surroundings been demonstrably in fault.

Season.—In a few instances where the time of year of the onset is mentioned it was in spring or summer, and in some others the time of year may be inferred as the parents have at first regarded the pigment spots as sun-freckles, and both Neisser and Vidal are inclined, therefore, to think that insolation may be an exciting cause in a congenitally predisposed child. These few facts are all we know of the etiology of the disease.

Symptoms.—When we review the symptoms and course of the disease we cannot but be impressed by the remarkable similarity of all the cases. Up to the time of the onset of the eruption all the parents state, that nothing

abnormal in the skin had been observable, nor were there any general symptoms to attract attention either before or at the time the disease began. Simply, the common history is that, some morning, probably in spring or summer, the face becomes covered with red spots something like those of measles, for which the disease has been mistaken, or the redness may be more diffused, forming patches. This erythematous eruption fades in a few days, but leaves behind pigmented freckle-like spots, or as in Duhring's, my own, and some other cases, the lentigines have appeared without any preceding eruption. Following at varying intervals of months or years, or it may be simultaneously, the other parts of the body which are habitually uncovered, viz. the neck, face, backs of forearms, and lower parts of arms, and sometimes the legs are similarly affected. When once the disease has attained to the dimensions exemplified by the cases shown to-night it ceases to spread, the further development being in the direction of fresh lesions within the self-prescribed limits. The pigment spots enlarge, become of a deeper colour, and others form, even in such unusual positions as the nails and palms, as in my cases, and the palpebral mucous membrane and sclerotic in one of Vidal's. As time goes on the pigment spots prove themselves by no means the harmless freckles they at first appear, but play an important part in the evolution of what are afterwards to become fatal lesions. We do not know how soon the white atrophic spots and areas develope but they are later than the pigmentation. And whether they are consequent upon the telangiectases, as Taylor thinks, or whether the telangiectases are the consequence of the obliteration of the vessels in the atrophic areas, remains to be proved. The atrophy sometimes involves the mucous membranes. At varying intervals, but usually some years from the commencement of the disease, superficial ulcers develope, covered with yellowish brown, or black blood-crusts. Some of the ulcers heal while others spread and fungate. A little later when the finger is passed over

the skin small verrucose projections can be felt situated upon the pigment spots. These warts are in most cases the starting-points of the tumours. Here I would call attention to a point not noticed before, namely, the prevalence of the ulcers and tumours upon the right side of the face. Out of twelve instances in which the side where these lesions began is mentioned, in nine it was upon the right and in only three upon the left side, and when both sides were involved they usually predominated upon the right.

Clinically, the tumours may be described as verrucose and fungating, but pathologically, all observers except Taylor, of New York, have described them as epitheliomas, with the epidermic glands abundant in some but absent in others. That they are not always or at all times epitheliomas the present case demonstrates, and Taylor's was not improbably like mine. It is, however, quite possible that if these tumours were left to irritate and be irritated that epithelioma might supervene, for the boundary dividing papilloma from epithelioma is often a very narrow one. Leloir and Vidal thought that the epitheliomas develop in other structures besides the warts, as they found nodules of epithelioma in the middle of the corium, which they thought originated in the glands of the skin. It must be borne in mind, however, that on the Continent they use the term epithelioma in a somewhat wider sense than we do. With regard to the production of the ulcers I believe them to be due to infective pus. I have seen some of them evidently produced by the discharge from the eye, and in such ill-nourished tissues they are easily excited and kept up. The fact that in these cases they followed in the younger children so soon after their appearance in the elder suggests a similar explanation. If untreated they tend to fungate and perhaps become the site of an epithelioma, while some have described them as becoming rodent ulcers.

The appearance of the tumours marks the beginning of the end. Glandular implication and general infection are

rare, though it probably occurred in Hebra's first patient, who died of cancer of the peritoneum when twenty-seven years old; but as the tumours multiply and fungate the health of the patient, which has hitherto been so remarkably good that no general symptoms belong to the disease, gives way at last from the continuous discharge and he dies marasmic and exhausted.

Vidal divides the disease into three periods:

1. The onset and development of the erythema and pigment spots.

2. The atrophy of the skin with its consequent dryness, wrinkling exfoliation, and accompanying telangiectasis and superficial ulcers.

3. The formation of verrucous projections whence the fungating tumours develop.

Diagnosis.—Few will dispute that the disease is *sui generis*, but errors may arise in its diagnosis. At the commencement the red spots have been mistaken for measles and the pigment spots for ordinary freckles; time and the surrounding circumstances will clear this up. Later on difficulties have arisen from laying too much stress on one lesion to the exclusion of the rest; thus the telangiectases have led to its being regarded as a rare form of nævus. From the scabbed ulcers and cicatricial aspect it has been pronounced to be a form of lupus; but what was it before the ulceration commenced, and what kind of lupus begins as superficial sores scattered irregularly over the face and without evident preceding infiltration of the skin?

But the disease that most closely resembles it is the atrophic stage of scleroderma universalis. Here we have immobility, white atrophy, telangiectases, and sometimes pigmentation, but in scleroderma the immobility is much greater, the telangiectases are rarely so conspicuous, and the pigmentation is not in lentiginous spots but in streaks, while the early history is totally different.

Indeed, error is almost impossible if the existence of the disease is borne in mind and all the lesions and the history of their development known.

The *prognosis* is gloomy enough. It is true that cases have gone on to adult age, and one which began late, viz. at nine years old, lasted for thirty years before the tumours made their appearance, but the majority succumb long before this and many have died under puberty. There is absolutely no hope of spontaneous improvement.

Treatment.—Everything yet tried, such as arsenic, tonics, cod-liver oil, and so forth has been absolutely futile. The removal of the tumours as a palliative is an obvious indication, and my experience of these three cases leads me to believe that much may be done for their relief by local treatment. The ulcers have nearly all been healed or are progressing in that direction. The recent ulcers have been treated with a weak ammonio chloride of mercury ointment, followed by boracic acid ointment. The old indolent or fungating ulcers were first scraped with a sharp spoon and then dressed with boracic acid lint or ointment. The eyes, under Mr. Tweedy's direction, have been diligently bathed with boracic acid lotion and greatly improved, and thus fresh ulcers from the irritation of the discharge on the cheeks has been avoided. Those who saw the cases in January will bear witness to their improved appearance, and though no doubt this is only temporary, constant watchfulness and early treatment of fresh lesions would almost certainly delay the progress towards death by saving the strength of the patient.

Pathology.—As to the general nature of the disease all Continental authorities are agreed that it is an atrophy of the skin, probably due to a neurosis, which we can only ascribe to a congenital predisposition, though all do not agree that the atrophy is primary.

Granting the existence of a trophic neurosis many of the symptoms are not without analogies in other diseases:

Excess of pigmentation, followed by total absence of it, is often seen in leucoderma, and probably pigmentation always precedes the white patches in that disease. As a degenerative change we see pigmentation in elderly people, and at the present time I have two cases of old persons

where, after removal of eczema, pigmentation has been left which in one of them is distinctly freckle-like. Atrophy with pigmentation and telangiectasis, as before remarked, is seen in scleroderma, but here infiltration of the tissue precedes the atrophy, the telangiectasis, being, probably, due to collateral hyperæmia consequent upon the blocking of neighbouring vessels by the infiltration. The ulcers appear to be accidents so to speak and to a great extent avoidable, but the warts which are the starting-point of the tumours are probably another evidence of degenerative change analogous to the pigmented warts so frequent in senile atrophy of the skin, and these warts too are not unfrequently the seat of cancerous changes. Kaposi and Neisser explain the production of epithelioma at so early an age as due to the active changes going on in the epithelial tissues, in the production and rapid disappearance of new papillæ and epithelium and of the pigment-producing elements. The disease appears to me best explicable by regarding it as one in which the nutrition of the tissue is damaged in the same direction as in senile decay, and therefore prone, like worn-out tissues generally, to resent irritation by the production of morbid and too often malignant growths. The pterygium, a condition rarely seen in the young, is another symptom of this premature sensibility. I certainly prefer the above analogy to the other put forward by Vidal, who asks:

"May it not be a variety of that numerous class of epithelial cancers of which the first and second stages are only the prelude, and the disease is only established at the third period?

"In superficial epitheliomas from multiple foci, which in various forms we call benign epitheliomas, the cancroid may be preceded for years by a dry seborrhœa or an epidermic exfoliation of a greyish or brownish colour. Gradually this lesion passes insensibly into a cancroidal ulceration which, superficial at first, ultimately gets deep. Compare also the affection of the tongue which, after lasting perhaps twenty years, becomes cancerous, the epithe-

lium having gradually got thick and rugose, this tylosis being the first stage of the cancroid."

One word about the name. Xeroderma is certainly an unfortunate selection, as that term is already in thoroughly established use for the mildest form of ichthyosis, with which this disease has nothing to do, and it can only therefore lead to confusion. Taylor's name, "Angioma pigmentosum et Atrophicum," depicts several features of the disease, but is too clumsy an appellation. The disease is admittedly an atrophy of the skin, and I would therefore suggest "Atrophoderma pigmentosum," which would call attention to two prominent symptoms, avoid confusion, and not be too complicated for ordinary use.

Table of Cases of Xeroderma Pigmentosum up to January, 1884.

No	Age.	Sex.	Consanguinity.	Date of onset.	Age when tumours first appeared.	Nature of tumours.	Observer.	Reference.	Remarks.
1	18	F.	—	Early childhood	?	—	Hebra and Kaposi	Hebra's Dis. of Skin, Syd. Soc., vol. iii, p. 254	Died of cancer of the peritoneum, æt. 27.
2	10	F.	—	„	11 years	Epithelioma	„	Do.	Died of cancerous cachexia, æt. 13.
3	8	F.	2 sisters	1 year	8 years	Sarco-carcinoma	Geber and Kaposi	Do.; also Archiv Dermatologie und Syphilis, 1873, Heft i	Tumours began on nose, probably on right side, as it was worse there.
4	6	F.	2 sisters	„	None	—	„	Do., do.	Geber called it a rare form of nævus.
5	5½	F.	Sister and brother	2 years	„	—	Kaposi	Medizinische Jahrbücher, 1882, p. 619	Had meningitis when 6 months old.
6	2½	M.	Sister and brother	„	„	—	„	Do.	Not seen by Kaposi; described by parents.
7	17	M.	—	?	12 years	Epithelioma	„	Do.	First tumour on right cheek; became very numerous, and were destroyed.
8	22	M.	—	16 years	20 years	„	„	Do.	First tumour on left lower eyelid; numerous ulcers elsewhere, the first on right ear.
9	?	F.	3 sisters	Very early life	—	Angio-myxoma	R. W. Taylor	Abstract in Archives of Dermatology (American), vol. iv, pp. 66, 343	No details given.
10	?	F.	3 sisters	„	—	„	„	Do.	
11	?	F.	3 sisters	„	—	„	„	Do.	
12	?	F.	2 sisters	„	—	„	„	Do.	
13	?	F.	2 sisters	„	—	„	„	Do.	
14	?	F.	—	—	—	—	„	Do.	
15	?	F.	—	—	—	—	„	Do.	
16	40	M.	—	9 years	About 39 years	Epithelioma	Heitzmann	Do., vol. iv, p. 67	First tumour on left cheek.

17	17	F.	—	6 months	None	—	Duhring	American Journ. of the Med. Sciences, vol. lxxvi, p. 424	Mother died of cancer of uterus. Has not altered from age of 9 years; scalp was invaded; freckles over the first lesion.
18	5 mos.	M.	7 brothers	5 months	„	None	W. Rüder	Rüder, Ueber Epithelial-carcinom der Haut bei mehreren Kindern einer Familie, Berlin, 1880 (quoted by Neisser in Viertelj. für Dermat. und Syph., 1883, p. 51)	Skin rough and flabby on neck and arms only; no mention made of pigment.
19	18 mos.	M.		—	„	—	„		Many small warts, pin's-head size, on arms.
20	4	M.		—	„	—	„	Do.	—
21	5	M.		—	About 5 years	—	„	Do.	Had a tumour on lower lip, which dropped off some weeks before he came under observation. Only scar left.
22	9	M.		—	8 years	Epithelioma	„	Do.	Tumour on lower lip, excised eight months before, recurred; warts very numerous all over affected area.
23	11	M.		—	11 years	—	„	Do.	An ulcerating wart beginning on right cheek.
24	10	M.		—	About 9 years	—	„	Do.	Extensive cancerous growth in temporal region was the cause of death.
25	24	M.	2 brothers	3 months	22 years	Epithelioma	A. Neisser	Viertelj. für Dermatol. und Syph., 1883, p. 55	Father died of obstruction of œsophagus. ? cancer. Began with red patches after a definite exposure to sun. Ulceration, which became epitheliomatous, began on right lower lid.
26	22	M.		„	None	—	„	Do.	Not seen by Neisser. Said to be exactly the same as brother, but no tumour.

No	Age.	Sex.	Consanguinity.	Date of onset.	Age when tumours first appeared.	Nature of tumours.	Observer.	Reference.	Remarks.
27	11	F.	2 sisters	3 years	9 years	Epithelioma, fungoid	E. Vidal	Annales de Derm. et de Syph., vol. iv, No. 11	Tumours began on right side of nose.
28	11	F.	2 sisters	18 mos.	4½ years	,,	,,	Do.	Tumours began on right cheek, and others followed on same side. Died at the age of 11 from exhaustion.
29	15	M.	3 brothers	2½ years	4 years	Epithelioma, verrucoid	,,	Do.	Corneous vegetation on root of nose, right cheek, and upper lip.
30	12	M.	3 brothers	2 years	4 to 6 years	,,	,,	Do.	Most of tumours on the right cheek; had one which dropped off on right hand.
31	10	M.	3 brothers	,,	5 to 7 years	,,	,,	Do.	Right side of nose showed first tumour; others followed on left side and median line of face; several dropped off. Died, æt. 10 years.
32	12	F.	2 sisters and their brother	Began between 12 and 18 months	10½ years	Papilloma, fungoid	H. R. Crocker	—	All the large tumours are on the right side of face, and most of the sores also.
33	10	F.	2 sisters and their brother	Began between 12 and 18 months	Sores at 4yrs.	None	,,	—	Has many crusted sores, but no fungous tumours yet; nearly all on right side.
34	9	M.	2 sisters and their brother	Began between 12 and 18 months	Sores at 3yrs.	Papilloma, verrucoid	,,	—	Has many crusted sores, mostly on the left side. He had one tumour which projected more than 1½, became strangulated, and dropped off. The sores developed on the two younger children within one month of their appearance on the eldest, their respective ages being 6, 4, and 3 years.

84 cases.	From 5 mos. to 40 years.	17 males. 17 females.	9 families. 26 cases.	29 in the course of 1st 2 years.	From 4 to 39 years.				Of 14 cases where site of first tumour is mentioned definitely, 3 began in median line, 9 on right side of face, 3 on left.

Table of Doubtful Cases (or which have been quoted as Xeroderma Pigmentosum).

1	26	F.	—	22 years	None	—	Glax	Arch. für Dermat. und Syph., vol. ii, 1875, p. 114	This is clearly a case of atrophic scleroderma.
2	—	—	—	—	—	—	J. Hutchinson	Lancet, vol. i, 1875	Quoted by Neisser. Mr. Hutchinson reports it as a case of xeroderma of Wilson = Ichthyosis, which it undoubtedly is.
3	—	—	—	—	—	—	Tanturri	Il Morgagni, Juli, 1877	Quoted by Neisser. Called "idiopathic, pigmentary, telangiectacic, small-celled sarcoma of the skin." Have not seen original paper, but *primâ facie* it is not unlikely to be xeroderma pigmentosum.
4	—	—	—	—	—	Unilateral idiopathic cutaneous atrophy, with pigmentation and hypertrophy of cutaneous capillaries	Atkinson	Richmond and Louisville Med. Journ., Dec., 1877	Duhring, who has seen the original paper, considers this a case of morphœa, or closely allied to it.
5	28	F.	—	—	—	General atrophia cutis	E. Wilson	Diseases of Skin, 6th ed., p. 394	Began at 21 years. Closely allied to general Scleroderma.

DESCRIPTION OF PLATES III, IV, V.

Three cases of Xeroderma Pigmentosum (H. RADCLIFFE CROCKER, M.D.).

PLATE III.

Alice Elizabeth Beddoes, æt. 12 years.

PLATE IV.

Benjamin Beddoes, æt. 9 years.

PLATE V.

FIG. 1.—One of many lobed masses embedded in granulation tissue from a filbert-sized tumour on the cheek. × 100. Reduced one half.

a. Small epithelial cells filling the lobes.

FIG. 2.—Portion of the upper part of a small ulcer from the cheek.

b. Lowest layers of horny part; the rest has been excoriated.

c. Dilated vessels.

FIG. 3.—A single lobe of the papillomatous tumour. × 350.

Mintern Bros. Chromo lith.

Mintern Bros. Chromo lith.

Fig 1.

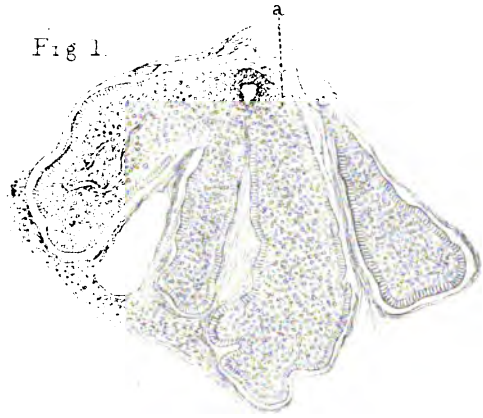
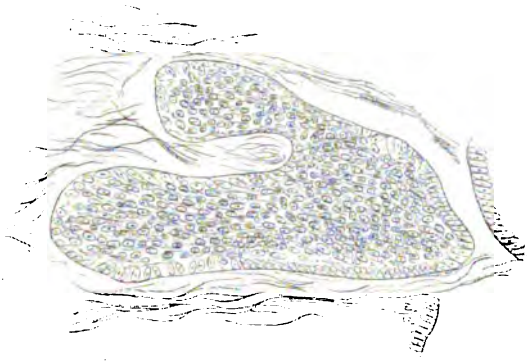


Fig 2.

Fig 3.



THE PATHOLOGY OF MYXŒDEMA

AS

ILLUSTRATED IN A TYPICAL CASE.

BY

JOHN HARLEY, M.D. Lond., F.R.C.P.,

LECTURER ON GENERAL ANATOMY AND PHYSIOLOGY AND PHYSICIAN TO ST. THOMAS'S HOSPITAL.

(Received February 19th—Read April 8th, 1884.)

In his paper on this subject, published in the sixty-first volume of the 'Transactions' of this Society, Dr. Ord has given faithful reproductions of two photographs of Harriet Brown, one taken at the age of twenty-one when in a state of health, and the other at the age of twenty-seven when the subject of fully-developed myxœdema. She came under my care about three years before she was brought under the notice of this Society in 1877, and continued so up to the time of her death in 1881, and was regarded at St. Thomas's as a typical example of myxœdema. As I am now able to give a complete account of her case, I think it will prove interesting as a supplement to Dr. Ord's paper, and as an aid in the elucidation of this morbid condition.

At the age of twenty-one H. B— was a bright, active, slender woman, about 5 ft. 3 in. in height, and weighing

between eight and nine stone, of fair skin and dark hair. She had always enjoyed good health, there was no remembrance of any particular disorder in infancy, and she never had scarlet fever. Her general appearance at this time is given in Pl. vii, fig. 1, vol. lxi of the 'Transactions.'

Her parents, a sister, and two brothers are still alive, strong, and healthy. Her second and last child (who is now a bright, lively, spare, and healthy little girl) was born in 1871, at which time the patient was twenty-six years old.

The disease undoubtedly had its origin at this puerperal period.[1] The labour was not a good one, and recovery was protracted and imperfect. A year afterwards the myxœdema was fully developed, as will be seen in fig. 2 of the plate just referred to.

Three years later she came under my notice, and for the next five years, *i.e.* up to 1879, there was no noteworthy change in her condition. She merely complained of debility. The integument of the face was thickened, the eyelids puffy, and the bright crimson injection of the fair cheeks contrasted with the very dark brown hair. Excepting an occasional slight pitting over the tibiæ, and a little puffiness of the ankles, there was no thickening or œdema of the legs. All the functions were normally performed. The urine was frequently examined, and always found of rather low specific gravity and free from albumen. The pelvic and abdominal viscera were healthy.

The only defects discernible were deficient expansion of the chest walls with slightly prolonged expiration and impaired resonance—the usual indications of chronic pleuritic adhesions—and a rather feeble ventricular contraction. The pulse was correspondingly weak, but otherwise quite normal.

She took chalybeate tonics with advantage, and was always able to attend to her household duties.

Becoming a little weaker she was admitted into Christian Ward on 2nd September, 1879, and remained

[1] See p. 194.

in the hospital fourteen weeks. She said that three weeks previously she was suddenly taken with giddiness and weakness in the left side, and she should have fallen but for help.

The following was her condition at this time :—She was fairly well nourished, complained of increased weakness, of pain in the back of the head and down the back, of impairment of memory and of vision of the left eye. Excepting a little weakness of the left leg there was no special diminution of muscular power, nor was there any sign of nerve lesion or disorder. There was a little cough at night, a few bronchitic râles in the right lung, and a little crackling and diminished resonance at the left base posteriorly. The apex beat of the heart was neither visible nor palpable, but the position and sounds were normal; the pulse 72, weak. Both flanks were rather dull, but the abdomen was otherwise normal. There was slight œdema over the shins, and the feet became a little puffy on standing. The urine was normal and averaged about forty-eight ounces.

From the 9th to the 12th September the urine decreased to fourteen, twelve, and four ounces in the twenty-four hours, but continued normal; the puffiness of the face and œdema of the feet increased, the skin was moist, pulse 68, unchanged, temp. 96°, resp. 20. She was depressed, complained of great pain in the back, and had frequent nausea.

On 13th September and subsequently, the normal amount of urine, sp. gr. 1013 to 1017, was passed, but there was no improvement in the symptoms.

On the 15th she vomited, but the bowels continued regular, as was always the case.

During the rest of this month and the whole of the next she grew worse, and was wholly confined to bed, being occasionally restless, fretting over imaginary troubles, and getting out of bed, and in doing so on one occasion she fell and bruised herself. She was alternately drowsy and restless with slight delirium, muttering to herself and

being frequently under the delusion that someone was waiting for her. She complained of sick headache; the pulse was small and weak, but not accelerated; the heart sounds very feeble and hardly to be heard; the temperature from 1° to 3° subnormal (see below). The urine varied from twenty to thirty-six ounces, sp. gr. 1007 to 1017, occasionally depositing a slight amount of lithates; on two or three occasions it was darker coloured and deposited phosphates. The amount of urea excreted at intervals was as follows:

Oct. 30	... 24 hours	... 140·5 grs.			
Nov. 6	... „	... 90·5 „	...	Sp. gr. of urine 1013	
„ 8	... „	... 101 „			
„ 13	... „	... 196·6 „	...	36 ounces of urine	
„ 18	... „	... 66·9 „	...	34 „	„

The average for the five days is 119·1 grains, being a reduction to about half the amount excreted by a healthy individual of her sex, size, and weight.

During the time she remained in bed and for some time after, the temperature was carefully recorded night and morning at the same hours; it was constantly subnormal from 1° to 3°.

Average for the 1st five days,		Aug. 31 to Sept. 5	96·2° Fahr.			
„	2nd „	Sept. 6 to „ 10	96·2° „			
„	3rd „	„ 11 to „ 15	97·4° „			
„	4th „	„ 16 to „ 20	96·9° „			
„	5th „	„ 21 to „ 25	96·7° „			
„	6th „	„ 26 to „ 30	96·3° „			
„	7th „	Oct. 1 to Oct. 5	95·9° „	}	Constantly in bed.	
„	8th „	„ 6 to „ 10	96·3° „			
„	9th „	„ 11 to „ 15	96·7° „			
„	10th „	„ 16 to „ 20	95·6° „			
„	11th „	„ 21 to „ 25	96·6° „			
„	12th „	„ 26 to „ 30	95·7° „			
„	13th „	„ 31 to Nov. 4	96·3° „			
„	14th „	Nov. 5 to „ 9	96·6° „	}	Began to sit up in the afternoon.	
„	15th „	„ 10 to „ 14	95·8° „			
„	16th „	„ 15 to „ 19	96·6° „			
„	17th „	„ 20 to „ 24	97·7° „			

The highest temperature recorded is 97·7°, and the difference between this and the lowest was only 2°.

On the 7th November she was nearly relieved of the cerebral symptoms, but she still felt weak and giddy, and had a little pain in her limbs; she walked slowly, moving the limbs equally. The pulse standing was 92 and regular, but the slightest pressure effaced it.

On the 15th of the same month she had returned very nearly to her usual state, eating and sleeping well. Five days before she left the hospital (December 12th) the catamenia reappeared after an absence of a year.

After this illness there was a marked declension of strength, but she still got up early in the morning and attended to her household duties, her condition remaining very uniform for the next two years nearly.

On the 30th September, 1881, she was again admitted into Christian Ward under my care, and died five days afterwards. She complained of loss of power in the left side so that she could not walk without help; she had a troublesome cough and a little bronchial expectoration. Her appearance and manner were unchanged, except that the expression was heavier and the speech thicker and more laboured. The skin was dry and harsh, that of the chest being rough with raised papillæ. The respiration was almost entirely abdominal, 24. The breath sounds were normal on the right side excepting at the apex and down the back to the side of the vertebræ; here it was rather harsh and the expiration was prolonged. The left chest was dullish everywhere; there was an increase of vocal fremitus and bronchophony, and the breath sounds were rather tubular. The apex beat of the heart could not be either seen or felt, the first sound was inaudible, the second clear and sharp; pulse regular, small, and feeble, 72. Tongue clear and moist, appetite defective, bowels regular, the abdomen normal, the urine normal in quantity, sp. gr. 1020, free from albumin. The catamenia had been absent three months.

Left to herself she lapsed into the drowsy, semi-delirious

state above described as her condition when in the hospital nearly two years previously. The highest temperature recorded was 97·4°, but the four last days of her life it was only 94°. There was retention of urine the second day after admission. The day before her death she could be roused, and passed water without assistance. She died of asthenia.

The post-mortem examination was made by Dr. Gulliver. The body was well nourished, the skin singularly fair, like alabaster or œdematous tissue, but quite free from œdema excepting around the ankles, where pressure caused slight pitting. There was a moderate amount of pure white adipose tissue. The peritoneum contained a pint and a half of clear serum; here and there there were a few old fibrous adhesions, but about the uterus and ovaries they were strong and general.

The *liver* weighed 2 lb. 13½ oz. On the surface a few scattered yellow dots—not raised—were observable, but the structure was normal.

The *kidneys* weighed together 9½ oz.; they were lobulated, slightly elongated, and narrow. The section was unusually firm and glistening, the cortex injected, the capsule stripped off too easily, and the arteries were thickened. Otherwise the organs were quite healthy.

The *uterus* was nearly twice its normal size, but otherwise healthy.

The *spleen*, *intestines*, and the rest of the pelvic and abdominal viscera were normal.

The *tongue* was unusually firm and contracted; the *thyroid gland* reduced to two little yellowish-white masses, together about the size of the last joint of the little finger. The *chest* was narrow and the muscles pale and stringy. The *pericardium* was distended with clear straw-coloured serum; there was no evidence of inflammatory action.

The *heart* weighed 7½ oz., it was firmly contracted and empty, the muscle was healthy, and the valves also, and competent; there were a few patches of commencing atheroma at the root of the aorta.

The *right pleural cavity* contained half a pint of clear serum, the apex of the *right lung* (which weighed 1 lb. 1½ oz.) was firmly fixed by old adhesions; a cicatricial depression corresponded to the adhesion, and an inch internal to this there was an old nodular thickening with here and there a little cretaceous deposit. The posterior border of the lung was devoid of air and in a state of fibrous degeneration.

The *left lung* weighed 1 lb.; it was everywhere firmly adherent to the chest, and behind and at the sides the pleura was converted into a thick opaque fibrous membrane, and the lung could only be separated by dissection or laceration. The whole of the lung was in a state of fibrous degeneration, being very tough, completely free from air, and sank in water. Sections presented a streaky appearance due to the paler bronchial tubes and an increase of connective tissue around them. A teaspoonful of creamy odourless pus oozed from one of the divided bronchia, and the same amount from the trachea as it was divided opposite the supra-sternal notch.

The thoracic gangliated cords were deeply implicated in the adherent and degenerated pleuræ, and on the left side the ganglia could not be satisfactorily distinguished.

My colleague, Dr. Hadden, undertook to examine the *cervical sympathetic* and some other of the tissues. The following is his report:—"There is no alteration which can be pronounced abnormal in the cervical sympathetic; some of the ganglion cells appeared unusually large and pigmented, but on comparison with normal specimens, I could not satisfy myself that there was much variation from the healthy condition. But on comparing these ganglia with those of another case which I have recently examined, the similarity of the changes in the two cases is very striking. The lesion consists mainly in a sclerosis causing atrophy of the fibres, leaving the cells almost unchanged.[1] The changes in the skin are very marked.

[1] I have examined Dr. Hadden's specimens, and adopt the first six lines of his report, but hesitate to endorse the ninth and tenth. The blood-vessels appear

All the hairs seen in the sections are broken off short, none reaching to the surface. The fibrous layer of the hair sac is much thickened by a nucleated fibrillated tissue; the inner and outer root sheaths are obscured by a small round-celled growth, so that no normal structure can be seen. The coiled tubes which compose the sweat glands are filled with closely-packed small round cells, which become deeply stained by colouring matters; there is no lumen, and the polyhedral cells which normally line the interior are quite absent; the sudoriferous ducts near the sebaceous follicles are also blocked with similar cells; the adventitia of the small arteries seems unusually thickened. In one portion of the skin the fibrous bundles of the corium and superficial part of the subcutaneous tissue are separated one from another, but this appearance is probably artificial, for in most specimens no such condition is observable. The elastic fibres exhibit no change."

The brain and spinal cord were firm and apparently quite healthy. Apart from the morbid changes above detailed two main facts were obvious. Firstly, *the quantity of blood was small;* although the heart was contracted and empty, and one lung was degenerated and much less vascular than the normal organ, there was no noticeable fulness either of the superficial veins during life or of the great internal veins after death. Secondly, *all the tissues were abnormally firm and tough:* there was a general increase of connective tissue throughout the body. This was well illustrated in the muscles. For the purpose of the chemical examination of the tissues, the results of which are given below, I procured a new "Nye's mincing machine." It minced ordinary flesh readily and perfectly, but on trying to pass the rectus abdominis muscle of the patient through it, the knives became

to be wider and more numerous than usual. It would be interesting, of course, to find such widespread implication of the sympathetic in these cases, but I think that the symptoms in the case under consideration are sufficiently accounted for by the involvement obvious to the naked eye, of the *thoracic ganglia.*

entangled in tough strings of fibrous tissue and the instrument was so much a failure that I had to resort to the old plan of "the chopper and block."

CHEMICAL EXAMINATION OF THE TISSUES.—My object was simply to determine the amount of mucin. The process which I adopted was as follows:—One part of the tissue (15 grammes=238 grains) was pulped or minced and digested for a week with frequent agitation in seven parts (107·9 cubic centimètres = 1666 grain measures) of fresh lime-water at 60° Fahr., and then filtered. The residue was then macerated in a fresh portion of lime water for from four to eight hours at a temperature gradually rising from 60° to 110° Fahr. At the end of this operation the softer tissues were completely disintegrated. The solutions were then filtered, and the mucin both of the first and second filtrate precipitated by acetic acid, collected upon a filter (associated with a second of equal weight made from the same sheet of paper), washed with water acidulated with acetic acid, and estimated in a dry crisp state. In the second maceration it is obvious that if chondrin were contained in the tissue, the amount would be included in the figures representing the amounts of mucin. But it may, I think, be assumed to be absent from all the tissues examined except the lungs. In the estimation, however, given below no artificial heat was employed, the second maceration (with heat) giving no appreciable precipitate with acetic acid.

The following is the result:

Percentage of Mucin in the following Tissues of Harriet B—.

Skin	0·525
Sheath of rectus	1·239
Subcutaneous fat of abdomen	0·176
Muscle	None
Lung[1]	1·600
Kidney[1]	0·804
Spleen	None

[1] I regret that I failed to note the parts of the organs from which the

I am not aware that the proportions of mucin and the other constituents of healthy tissues at various ages have been ascertained, and until this has been done, the value of the foregoing data is of course unknown. As a help to myself and future inquirers, I have determined this question to some extent in respect of a woman, Eliza B—, æt. 24, who died under my care in St. Thomas's Hospital. She was the subject of mitral disease. There was slight œdema of the eyes, and a little fluid in the serous cavities.

Percentage of Mucin, Albumin, and Gelatin in the following Tissues of Eliza B—, æt. 24.

	Mucin.	Albumin.	Gelatin.
Skin	0·17	0·68	19·50
Sheath of rectus abdominis muscle[1]	0·28	3·63	21·79
Subcutaneous fat	0·01	0·65	1·72
Muscle (rectus abdominis)	0·73	14·65	2·16
Lung (thin anterior border)	0·31	6·62	5·64
Kidney (cortex and medulla equally)	1·09	3·82	3·47
Spleen	None	Abundance	Abundance

The tissues, drained of blood and finely minced or pulped, were digested in seven parts of lime-water, and subsequently in a fresh portion at a temperature ranging from 110° to 160° Fahr.; the skin and tendon requiring the higher temperature in order to effect a complete solution. Indeed, all the tissues may be at once exposed to the higher temperature, when the process may be speedily effected. I used cold lime-water at first, thinking that heat might cause some decomposition of the mucin. A carefully performed experiment on bronchial mucus proved to me that mucin in lime-water undergoes no diminution when exposed for many hours to a temperature over 160° Fahr.

The process adopted was as follows:—Solution of the tissue having been effected, the filtrate was supersaturated

portions analysed were taken; the significance of this (see p. 199) occurred to me subsequently.

[1] The large quantity of albumin was no doubt derived from adherent muscular fibre.

with acetic acid, precipitated mucin was collected upon a filter (placed by the side of another which served as a counterpoise, being of equal weight and equally saturated with the solution, and washed and dried together), washed, and dried. The filtrate was next treated with solution of potassium ferrocyanide so long as any precipitate formed; the albumin was collected, washed with water acidulated with acetic acid, dried, and weighed. The filtrate was lastly precipitated by freshly prepared solution of tannic acid, and the gelatine collected, washed, and dried.

In ascertaining the amount of mucin it is necessary to state the part from which the specimen was taken; for example, the cortical part of the kidney will yield less mucin than the medullary portion, and the lining membrane of the pelvis more than the medullary portion. Again, with regard to the lung, it will be obvious that the thin edge of the lung will yield less mucin than the central parts, because a portion from the latter contains larger and more numerous bronchia than the former. The quantity of albumin, too, will vary with the amount of blood retained in the tissue.

The following observation shows this:

James P—, æt. 37, died under my colleague, Dr. Stone, of enteric fever and pulmonary congestion.

Percentage of Mucin, Albumin, and Gelatin.

	Mucin.	Albumin.	Gelatin.
Kidney cortex	0·064	4·34	4·34
„ medulla	0·64	4·30	6·19
Lung, diaphragmatic edge . .	0·42	0·43	8·42
„ root	3·43[1]	1·95	11·89

Taking the two analyses together, it appears that, of those examined, the organs which yield the largest amount of mucin are the lungs and the kidneys. This was of course to be expected since the mucous membrane freely extends into both of them.

[1] This large figure represents the combined amount of mucin and chondrin, by far the greater proportion being chondrin.

Further observations will determine whether or not the quantity of mucin in the tissues of the myxœdemic patient is in excess. Compared with the analysis in the other case, the total amount is nearly double, and so far, Dr. Ord's idea of a "myxœdema" is borne out. But it appears to me that the question of an increase of mucin sinks into insignificance in face of the unmistakably great increase of the connective or fibrous tissue, and to which the increase of mucin, if this be hereafter demonstrated, may be regarded as secondary.

The morbid condition in the myxœdemic case was associated with a general increase of the fibrous tissue at the expense of the parenchyma, and hence the integumental thickening and puffiness, and the increased hardness and toughness of the tissues. Such a case as that I have described might be appropriately defined as one of "*general fibrous invasion,*" producing a *chronic, cold debility.*

In this case, one lung was degenerate and useless, and as a consequence, the vital functions were proportionately reduced; the temperature was low and the urea reduced to half the normal amount. As a secondary effect, the parenchyma, adapting itself to the wants of the system, had decreased, and its place was supplied by connective tissue, which in process of time had become dense and inactive.

It remains to be considered how far the nervous system is involved in the general fibrous degeneration. In the case under consideration the cerebro-spinal system was evidently very slightly affected; the mechanism of thought, speech, and motion were more or less impeded, and there was a slowness of each proportionate to the general debility; the intellect, however, remained unclouded, and there was an absence of paralytic symptoms. But the sympathetic system is in many of these cases more deeply involved. Since these cases have come under my notice I have always referred them to degeneration of the sympathetic centres. A case of so-called "simple

atrophic sclerema," but really one of fibrous degeneration of the lungs and thoracic sympathetic, published by me in vol. lx of the 'Transactions' of the Society, is conclusive on this point; and the present case is as strong a corroboration as can well be offered. The myxœdemic and scleremic conditions in these patients were merely the consequence of severe internal inflammation experienced years before and affecting, *inter alia*, the pleuræ and peritoneum and the sympathetic ganglia adjacent to these membranes. It is easy to trace the effect of this, viz. a loss of tone in the blood-vessels of the connected viscera and a corresponding diminution of functional activity. The blood-vessels are the first organs to be affected by fibrous degeneration; the implication of the rest follows as a matter of course.

In the two cases to which I have just referred, fibrous degeneration of the lung was a notable factor in the disease, but it is of course conceivable that myxœdema (general fibrous invasion) or scleroderma and its allies may have their origin in inflammatory or other changes exclusively confined to the sympathetic ganglia.

If this view of the pathology of the disease be correct, myxœdema cannot be regarded as a specific disease, but merely as a variety of what I have termed "a chronic, cold debility" dependent on depression of the vital functions and frequently associated with fibroid degeneration of the lung, a condition usually, if not always, traceable to some severe antecedent illness attended by internal inflammation.

I am able to present to the notice of the Society to-night a patient of the male sex, who may be regarded as a typical example of myxœdema, and whose history and condition are strongly corroborative of the views here advocated. The following is a brief outline of the case:

George K—, æt. 52, a seaman, who has followed his profession until a few years ago. He is still a well-developed man, 5 feet 10½ inches high, weighing 14 stone, and well covered with fat; circumference of chest 37½

inches, of thighs 23¾, of calves 17¾. The ankles and insteps are very œdematous, and there is pitting over the tibiæ as high as the knee, and also of the sides of the calves on making hard and prolonged pressure. For the last month the face, which before was puffy, has been distinctly œdematous around the eyes, and the integument over the upper lids hangs down as semi-transparent sacs. The lips are hard and swollen, so as to impede labiation, the voice is gruff and monotonous, and the speech slow and hesitating. The cheeks are rosy, from chronic injection of the small vessels. The thyroid gland is evidently absorbed. The skin is cold and dry, and the extremities, especially the arms and hands, are quite rough with epidermal scales; the hands and fingers are thick and clumsy and the nails incurved, those of the forefingers completely covering the end of the pulp; the hair is coarse; the urine is normal in quantity, always free from albumin, sp. gr. 1013—1020, and acid.

He has been in the hospital under my care for the last six months, and his condition has remained unchanged, except that during the last month there has been a slight increase of œdema.

He complains of weakness and giddiness, weakness of memory, dimness of vision, and dyspnœa on the slightest exertion; his gait is slow and heavy. The pulse is 70, but very small and weak; the impulse of the heart is absent and the sounds scarcely audible, but there is absence of valvular disease; the expansion of the chest is very imperfect, there is a little dryish crepitation at the bases of the lungs; over the right upper lobe and the left upper lobe in front the expiration is audible and prolonged. The right chest is dull below the nipple line from front to back, the dulness being most marked at the side where it extends an inch above the nipple line; in every part of this lung the breath sounds are feeble, and there is marked increase of bronchophony and vocal vibration; there is neither cough nor expectoration, nor any history of previous bronchitis. His appetite is very moderate, and the

digestion is weak; he is frequently troubled with nausea and heartburn, and has occasional attacks of vomiting, a condition very like that which was so prominent in the case of scleroderma above referred to (see p. 201). The average temperature for August and September (forty-one observations) was 97·4°.

He was a remarkably strong man until 1854, when he had measles and smallpox. The latter illness occurred on board ship in the English Channel; he had no medical attention and was ill three months. Since this time he has not been strong, and subsequently he had an attack of dysentery lasting six months. Five years ago he had erysipelas of the head. He has always been subject to rheumatism of a subacute form, aching of the legs and arms. Two of his brothers have died of chronic heart and lung disease, a third of chronic lung disease, and a sister of "fits."

The patient has gradually and very slowly lapsed into his present state. He is not confined to bed, and can still walk a distance of a mile, but breathlessness is easily provoked by very moderate exertion of any kind.

During the last few weeks this patient has experienced an increase of the œdema of the legs, and the swelling of the lids almost occludes the eyeballs. He has lately suffered an attack of conjunctivitis and slight iritis of both eyes. It was doubtless of rheumatic origin. The œdema of the legs is now very great. This patient I have quite recently discovered was under Dr. Stephen Mackenzie's care in the London Hospital, and he has given an account of him in the last volume of the 'Clinical Society's Transactions' (vol. xvi, p. 260).

I have thought it would be premature to examine the few cases of myxœdema that have been published in reference to the views set forth in this paper. What appears to be needed at the present time is a full and complete history of the cases as they present themselves to us, and thus we shall acquire the facts necessary for generalisation.

One question in the pathology of myxœdema I have left

unconsidered. It is the relation of the thyroid gland to the disease.[1] I have noted the atrophy of the gland in chronic lung disease, especially fibroid degeneration, and I believe that a depression of the respiratory function will be found to be the usual associate of atrophy of the thyroid; the activity of the gland being associated with increased blood-supply as in the plethora which sometimes follows the natural cessation of the catamenia; while its atrophy may be expected when, by degeneration of one half of the respiratory surface, the blood is proportionately impoverished.

[1] See Dr. Stephen Mackenzie's paper, "On the Weight of the Thyroid Body," at the end of the volume.

AN

AVERAGE OR "COMPOSITE" TEMPERATURE CHART OF SMALLPOX.

BY

G. ERNEST HERMAN, M.B. LOND.,
M.R.C.P. LOND., F.R.C.S. ENG.,
OBSTETRIC PHYSICIAN TO, AND LECTURER ON MIDWIFERY AT, THE LONDON HOSPITAL, ETC.

(Received April 8th—Read May 27th, 1884.)

DURING the smallpox epidemic of 1871 I had charge, as resident medical officer, of a temporary smallpox hospital opened in Shoreditch. The nursing was superintended by sisters of St. Saviour's Priory, Hackney Road (a branch house of St. Margaret's, East Grinstead), and thermometric observations were taken and recorded by them. They were educated ladies who understood what was required and did it conscientiously, and I am satisfied of the accuracy of their temperature records. My colleague, Dr. Sutton, who acted as visiting physician to the hospital, permits me to say that he concurs with me in this.

From their observations, made under my own supervision, I have constructed the average or "composite" charts which are appended.

The first one is based on thirty-one cases, all of which recovered. In none of them was there any evidence of

vaccination, and in each of them the course and characters of the disease were those of smallpox unmodified by vaccination. In each case the temperature was throughout taken three times daily, morning, afternoon, and evening. The chart has been made by adding together the figures indicating the temperatures in the different cases for the same day of the disease and period of the day, and dividing the total by the number of observations forming it. The average temperature for each day of the disease and period of the day thus obtained has been set down on the chart. It does not begin till the third day of the disease, because none of the patients were admitted before that day; and the average temperatures for the early days are based on somewhat fewer cases than the rest of the chart, because some patients were not admitted till later in the disease than others.

So far as I can find no similar chart has been constructed before. The text-books illustrate the course of the temperature by specimen charts of individual cases. But in any individual case the temperature must be modified by circumstances special to that case. In an average chart such as this, the temperature variations due to the peculiarities of any individual case will be almost entirely neutralised by the larger number of other cases in which those peculiar modifying causes were not present. Therefore an average or "composite" chart, such as this, displays what may be called the typical temperature better than the chart of any individual case can do.

Chart II is intended to show the course of pyrexia in smallpox with the greatest possible clearness. It is simply Chart I with the slighter variations entirely neglected and the course shown in a diagrammatic form.

Chart III is based upon thirteen cases in which the temperature was taken every two hours for the period of twenty-four consecutive hours. These observations have been averaged in the same manner as those forming Chart I. In order to show the amount of variation from the average dependent simply on the time of day, as distin-

guished from that due to the different amount of pyrexia in the individual cases, the temperatures have been transposed, so that the lowest temperature in each case should stand at 101°. The dotted lines show the maxima of variation above and below the average.

Chart IV bears the same relation to Chart III as Chart II to Chart I, showing the diurnal fluctuation with the greatest possible clearness.

THERMIC CHARTS.

CHART 1.

Average, or "Composite" Chart of Smallpox.

The average temperatures, morning, afternoon and evening, of 31 cases of Smallpox unmodified by vaccination.

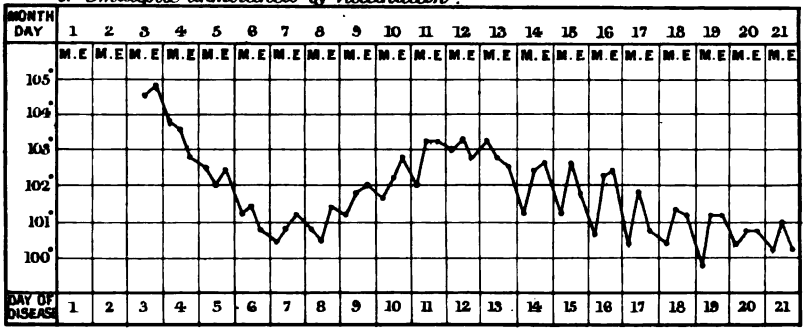
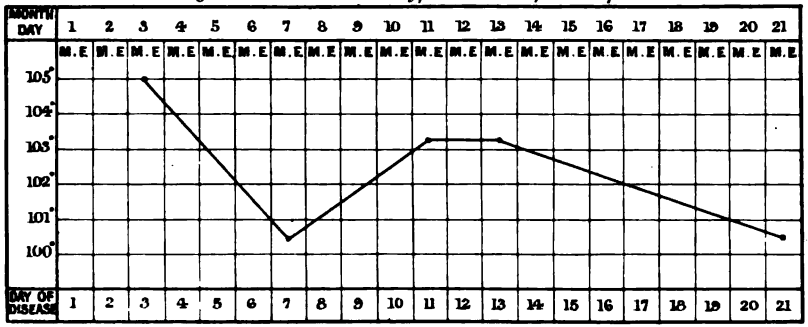


CHART 2.

Diagrammatic chart of typical smallpox temperature.

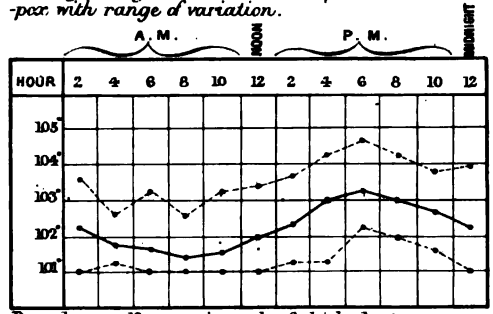


Mintern Bros. lith.

CHART 3.

(Period that of Secondary fever.)

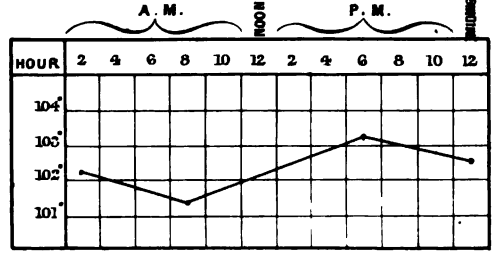
Average daily fluctuation of temperature in small-pox with range of variation.



Based upon 13 cases, in each of which the temperature was taken every two hours in the axilla, during 24 consecutive hours.

CHART 4.

Diagram of diurnal variation of temperature in smallpox.



A CASE OF GANGRENE OF THE LUNG

FOLLOWING

NECROSIS OF THE TEMPORAL BONE THE RESULT OF SCARLET FEVER.

TREATMENT BY DRAINAGE. RECOVERY.

BY

W. CAYLEY, M.D.,

PHYSICIAN TO, AND LECTURER ON MEDICINE AT, THE MIDDLESEX HOSPITAL
PHYSICIAN TO THE LONDON FEVER HOSPITAL, ETC.;

AND

A. PEARCE GOULD, M.S.,

ASSISTANT SURGEON TO THE MIDDLESEX HOSPITAL, ETC.

(Received April 8th—Read May 27th, 1884.)

Eliza W—, æt. 12, was admitted into the Middlesex Hospital under the care of Dr. Cayley on January 18th, 1884.

Her mother stated that she had always been a fairly healthy child, but four years ago had had an attack of scarlet fever and was for three months in the London Fever Hospital. On leaving there was a discharge from the left ear which continued for two years and then ceased. She appears to have been deaf with this ear since the scarlet fever.

A week before her admission into the Middlesex Hospital she was attacked by very severe pain in and behind the left ear and the discharge appeared again. A swelling

now formed over the mastoid process and she became very feverish and was delirious at night. There was no vomiting.

State on admission.—Pulse 96, resp. 26, temp. 100°. Patient is a pale, rather delicate-looking little girl complaining of great pain in the left ear. The left auditory meatus is blocked by granulations and exudes fœtid pus; over the left mastoid process is a tender œdematous fluctuating swelling, and on this side she cannot hear the ticking of a watch.

Pulmonary signs normal; there is a faint systolic murmur at the cardiac apex. Abdominal organs appear to be normal, urine free from albumen.

Chloroform was administered, and Mr. Gould made an incision over the mastoid process and let out about two ounces of pus. The whole mastoid process, together with the lower part of the squamous bone and the greater part of the tympanic ring, was found to be denuded, and there was a free communication between the abscess and the external meatus. A drainage-tube was passed through this, and the parts were irrigated with a carbolic lotion. The evening temp. was 103° (see Plate VII).

January 19th.—Temp. 101°; patient slept well and is free from pain, but is very irritable and cannot bear to have the ear touched. A little bloody discharge has issued from the drainage-tube. At 1 p.m. she had a severe rigor lasting ten minutes, after which the temperature rose to 104·6°.

At 3 p.m. she was again chloroformed and Mr. Gould trephined the mastoid process and with a small gouge removed some soft friable bone. She was now given ten grains of quinine, an ice bag was applied to the head, and at night a draught of bromide and chloral was administered. The next day the patient was free from pain and seemed better.

21st.—Temp. 97·8°, pulse 72; has slept well, is in no pain, no discharge from the wound. At 2.45 p.m. patient had a slight rigor lasting five minutes, after which the

temperature was 102·4°. She was given ten grains of quinine; the evening temperature was 98·6°.

During the next six days the patient seemed to be progressing favorably. She was almost free from fever, had no pain, and was able to take solid food; the wound looked healthy.

On Jan. 28th she became worse, she vomited, lost her appetite and became fretful and irritable. There was, however, no return of pain in the ear. The morning temp. was 97·6°, the evening 104°. She now felt burning hot and perspired profusely; her pulse was 150.

29th.—Has passed a restless night. To-day she is depressed and irritable. The temperature varied during the day from 97·8° to 103·8°; there was no pain in the ear.

30th.—At 2 a.m. this morning the temperature rose to 105·8°; ten grains of quinine were administered, and at 10 a.m. the temperature was 96·4°. Shortly after she had a severe rigor lasting ten minutes, and the temperature rose to 101·8°. She had a restless night, vomited twice, and now complains of pain on the left side of the chest. Ear is free from pain; at 10 p.m. the temperature rose to 103·2°; ten grains of quinine were given.

31st.—Was very restless and thirsty during the night and complained of severe pain in the head. This morning feels better. At 10 a.m. the temperature was 96·4°. In the evening she had another rigor, and the temperature rose to 103·2° and she vomited.

February 1st.—Is free from pain, but seems to be getting weaker.

2nd.—Temperature at 10 a.m. 104·2°. Has an irritable cough and complains of pain on coughing in the left hypochondriac and lumbar regions, and here there is a good deal of tenderness. The breath sounds on the left side are normal; but there is some dulness on percussion and feeble breathing at the right base. Ordered ten grains of quinine every night, cod-liver oil, and two ounces of port daily.

3rd.—In the night her nose bled; continues to cough.

4th.—Complains of pain on the right side, and fine friction sounds are audible in the axillary and mammary regions. At 1 p.m. the pain became extremely acute, and a subcutaneous injection of morphia was administered.

During the next three days little change took place in her symptoms, but she continued to get weaker and to lose flesh. Friction sounds continued to be audible, and the dulness at the right base became more pronounced; moist râles and bronchophony became audible. On February 7th it was noticed that her breath was offensive and she began to spit up small quantities of offensive pus. Since the regular administration of quinine at night the temperature, as seen by the chart, has not ranged so high.

11th.—It was noticed that there were bubbling râles on coughing over the right base; the dulness now extended as high as the angle of the scapula.

On Feb. 16th she had to be placed in a room by herself in consequence of the horrible fœtor of the breath and expectoration. Patient continued to lose flesh and strength and became emaciated to an extreme degree. She was constantly hawking up with great difficulty small quantities of fœtid pus, and it was evident she must soon succumb. After consultation with Dr. Powell I determined to have an opening made into the gangrenous cavity which we believed to be present in the base of the right lung. The physical signs were now absolute dulness on percussion as high as the angle of the scapula, bronchial breathing, and coarse almost bubbling crepitation at one spot below and internal to the angle of scapula.

On Feb. 23rd chloroform was administered and Mr. Gould introduced an aspirating trocar and cannula through the eighth right interspace about an inch external to the angle of the rib in a direction upwards and inwards. On withdrawing the trocar a few drops of fœtid pus escaped, and air passed in and out with the respiratory movements. A large-sized trocar and cannula were then introduced close to and parallel with the first, which was then withdrawn, but on removing the trocar no pus or air escaped,

showing that the cavity had not been entered, though the two trocars were close together. A piece of drainage-tube was then introduced through the cannula, which was itself withdrawn over the tube. A pad of boracic lint and oakum was placed over the side. In the evening patient was in great pain, still coughing and hawking up fœtid pus and perspiring profusely; pulse 136, very small and feeble; temp. 99°; given a subcutaneous injection of morphia.

24th.—Patient has passed a restless night and vomited several times. This morning a little fœtid pus escapes through the drainage-tube and air passes out on expiration.

25th.—Cough is better. She has ceased to expectorate. Dressings soaked with fœtid pus, and a dark stringy plug was withdrawn from the drainage-tube. This on microscopical examination was found to contain elastic fibres.

26th.—General condition improving. No expectoration. Appetite better. Dressings soaked with brownish, offensive pus.

27th.—Discharge abundant; less offensive. No expectoration. General condition continues to improve. Is very hungry, and was given at her urgent request pork chops and mashed potatoes. Breath much less offensive. Eats heartily. Discharge also less offensive.

Patient now continued steadily to improve and rapidly regained flesh.

On March 1st Mr. Gould changed the tube for a shorter one.

3rd.—The discharge was much diminished and no longer offensive; air still continued to pass in and out. To-day the tube was removed. There is still a discharge from the auditory meatus and from the wound over the mastoid process.

6th.—Patient able to get up. Appetite voracious.

8th.—Air no longer escapes through the opening. Discharge scanty and sweet.

13th.—Opening into chest is quite closed. Wound covered with healthy granulations and cicatrising at edges.

On March 27th she was sent to the Eastbourne Convalescent Hospital. The wound had nearly healed. There was impaired resonance over the right base with slight sinking in of the chest wall; the breath sounds were feeble but vesicular in character, and free from adventitious râles.

She was seen again on her return from Eastbourne and was then quite well. The wound had entirely healed.

Remarks.—In this case there can be little doubt but that the gangrene of the lung was the result of a pyæmic embolism, as the general signs of pyæmia, repeated rigors, fever of a markedly remittent type, and prostration were well marked.

The mode of treatment adopted was the only one which afforded the slightest hopes of saving the child, as she was evidently rapidly sinking from the effects of the pent-up gangrenous matter in the lung. And I believe that wherever we have to deal with a condition of this nature the case should be treated according to the well-established principles of surgery, and evacuation effected.

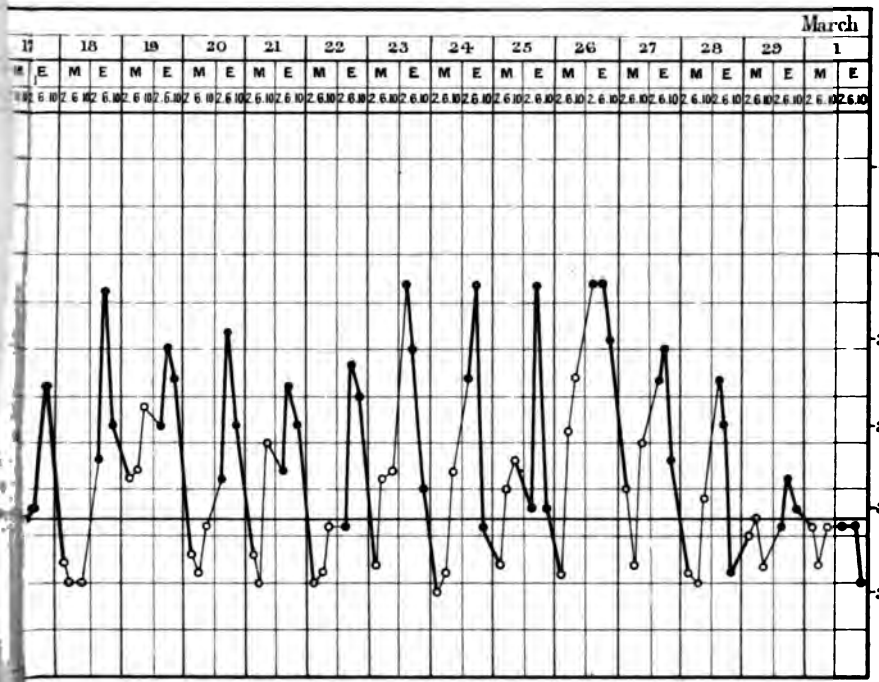
In 1879 I brought before the Clinical Society a case of gangrene of the lung which was treated by incision and drainage, and though here the patient was too far reduced to allow of his rallying, nevertheless the most marked relief to the distressing symptoms was obtained.

Sometimes, no doubt, a circumscribed gangrene of the lung causes but little constitutional symptoms, and the patients ultimately expectorate the contents of the gangrenous cavity and recover. But wherever the condition produces marked hectic or septicæmia, the operation should if praticable be had recourse to.

Where the pleura is adherent and the lung surrounding the gangrenous cavity solidified I believe there is no danger attending the operation. And where the gangrene is of some duration this condition is most likely to be met

with. And in all cases an exploratory puncture with a fine trocar may be made without much risk.

If there are no pleural adhesions it may perhaps be doubtful whether it would be right to incur the risk of converting the case into one of pyo-pneumothorax, though I believe even this condition would afford in many cases better chances of recovery than one of gangrenous septicæmia.



lutely unjustifiable. But there are others [illegible] different conditions obtain, and dictate the measures to be followed. For when the discharge, by its abundance

ON THE TREATMENT

OF

PUS-SECRETING BASIC CAVITIES OF THE LUNG

BY THE METHOD OF

PARACENTESIS AND FREE DRAINAGE.

BY

CECIL Y. BISS, M.A., M.D. (CANTAB.), M.R.C.P.,

ASSISTANT PHYSICIAN TO THE HOSPITAL FOR CONSUMPTION, BROMPTON, AND THE MIDDLESEX HOSPITAL.

Received April 8th—Read May 27th, 1884.

THERE are, perhaps, few questions in the practical treatment of pulmonary disease which are more interesting and more responsible than those which concern the course proper to be followed in certain cases of basic lung cavities, from the walls of which an abundant purulent secretion continuously exudes. Doubtless there are many instances of this condition in which the state or age of the patient, the comparative mildness of the symptoms, or other qualifying circumstances, place it beyond question that, whatever treatment may be adopted, surgical interference is absolutely unjustifiable. But there are others in which very different conditions obtain, and dictate the measures to be followed. For when the discharge, by its abundance

and fœtor, harasses, disgusts, and steadily enfeebles the patient, destroying his appetite, and draining blood and tissues of essential albuminoid elements; or when a febrile condition is induced with hectic symptoms and loss of flesh and strength; or, again, should pyæmic indications appear, especially metastatic suppurations, it is easy to discern that a limit has been reached beyond which abstention from surgical measures for the evacuation and free drainage of the cavity —whatever risks may be involved—will be inevitably fatal.

There is, however, a third class of cases of this nature, in which the physician's interest and responsibility reach their climax—a class which, in its clinical aspects, the severity of the symptoms, the constitutional proclivities and possibilities of the individual patient, and the bearing of other important particulars, lies intermediate between those already recited in which the operation of paracentesis is either forbidden or compelled.

In the cases of this intermediate class, the prospects of success by operative measures are sufficiently hopeful to warrant the risks incurred by the operation. This being so, it is certainly an inquiry of the highest importance whether such cases can be differentiated from others, and by what signs they may be distinguished and classified. For, it must be remembered, that while there are certain acknowledged risks involved in the puncture of the lung through the chest wall for the evacuation of the cavity, fatal results cannot be averted by inaction when continued suppuration is proceeding; and, on the other hand, valuable time and chances may be lost. If, therefore, it be possible to determine by a discussion of the elements of diagnosis and prognosis existing in this form of disease, and a comparison of the results of operations, what facts and principles are to guide to the operation of paracentesis, a valuable position in practical medicine will be reached.

With this view I venture to bring before the Society a case of basic cavity belonging, I conceive, to the third of the classes mentioned, in which paracentesis of the lung was decided upon. It is that of a patient who came under

my care at the Hospital for Consumption, Brompton, in November of last year, with a series of cavities in the lower part of the right lung. The operation was successfully performed with great relief to his symptoms, but he died eventually of pyæmia.

J. L—, a resident of Kensington, æt. 32, a letter-carrier by calling, was admitted on October 3rd, 1883, into Corry Ward, under the care of Dr. Reginald Thompson, by whose kindness I am permitted to bring the case forward, and to state that part of its history which transpired under his observation. The family history was rather indefinitely given, but the patient's father was living and healthy, as also were three brothers and four sisters. His mother had died of "bronchitis:" one brother had died as an infant from some unknown cause.

His own previous history was unimportant. He had "never had a day's illness in his life;" and, though following a calling which exposed him to all weathers, he had not suffered from coughs. There was no reason to suppose he had ever been intemperate, nor that he had ever contracted syphilis.

His illness appeared to have commenced six months before admission. He "caught a severe cold" and was obliged to give up work, but had not been confined to bed. A troublesome cough had come on and grown worse, attended by copious expectoration, and the sputa had been noticed to be slightly offensive. He had suffered from night-sweatings, shortness of breath, and progressive loss of flesh. A month after his illness began slight hæmoptysis appeared, and for a fortnight the sputa contained more or less blood, but never very much. He had grown gradually worse, and eventually sought admission to the hospital.

The note of his condition made at the first examination describes him as a man of middle height and spare build, considerably wasted, of a waxy pallid complexion, and with a dull worn-out expression. His breath was short, cough troublesome, and expectoration profuse and fœtid; the

tongue was thinly furred, appetite lost, and he felt too weak to care to leave his bed. The bowels acted regularly, and he had never suffered from diarrhœa; the urine was normal in every way.

He had no physical signs except those found in the thorax. The chest was narrow, but fairly well formed. The left lung was free from any morbid indications, and its breath-sounds were rather exaggerated. The condition of the right lung is thus described in a brief note made

FIG. 1.

by Dr. Thompson:—"Bulging in scapular region and below. Absence of fremitus and total absence of breath-sounds; marked dulness of percussion (over that area). Dilated lung in front. Heart's apex outside nipple-line" (see Fig. 1).

With this history, and these physical signs, the case was regarded as that of an abscess situated near the posterior base of the lung, and accompanied by a slight effusion into the adjacent pleura. In the formation of this diagnosis great stress was naturally laid upon two points: (1) The total absence of breath-sounds from the affected base; and (2) the displacement of the cardiac apex, which was felt

distinctly, though feebly, at a spot a little to the left of the nipple-line. The concurrence of these signs was regarded as evidence of an accumulation of fluid either in the pleural sac, or in the lung itself; and in order to clear up the point, a fine needle connected with an ordinary hypodermic syringe was introduced at the mid-axillary line in the sixth space, but with a negative result. A week later another exploration was made, with a somewhat larger needle, in the ninth space below the angle of the scapula. A drachm of pure blood was withdrawn but no pus, and a little air bubbled out through the needle, a little hæmoptysis occurring at the same time. It was concluded that there was no fluid in the pleura, that the lung was probably adherent to the parietes of the thorax, and that the needle had passed into the lung itself.

As the patient had slightly improved since his admission, his cough being rather better and expectoration less, it was decided to give him the full benefit of medicinal means before resorting to operative measures. He was accordingly directed to use an inhalation of iodoform through a respirator, and, a little later, he was placed on three grains of the Pil. Picis every six hours. The note made of his chest on the 10th October is: "There is complete dulness behind on the right side, with absence of respiratory murmur below the sixth rib; above that the respiratory murmur is weak. General surface friction audible in dorsal region. In front, chest is hyper-resonant." This absence of respiratory sounds over the posterior dull area is remarkable in the light of what follows.

A week later (17th October) his general condition had not altered much, but the temperature had risen, ranging from 101° to 102°. He was bringing up much expectoration, but it was less fœtid. The physical signs were, however, materially changed, for over the dull posterior area there was now audible marked cavernous respiration, with bronchophony and pectoriloquy; and after cough small crackling crepitation within the same limits. A week later these signs were unaltered.

On the 15th November the charge of the case devolved upon me in Dr. Thompson's absence; and upon making an examination I found the chest in the condition just described, with the addition of a loud friction-sound over the right axilla, where much pain was felt at the time. I was particularly struck by the following points:

1. That the left lung was perfectly free from morbid signs.

2. That the cardiac displacement persisted, the apex beating very distinctly about an inch outside its normal site, and the superficial cardiac dulness extending to the same point.

3. That there was no evidence of disease at the *apex* of the right lung, the breath-sounds though weak being normal, the percussion note unaltered, and no râles audible.

4. That over the dull posterior area where loud cavernous breathing was still heard, the râles had disappeared.

On the 5th December another exploration was made with a rather larger aspirating needle than that previously used, the puncture being made in the eighth space below the angle of the scapula, but no fluid was obtained. The needle passed deeply inwards and seemed to penetrate a solid resisting mass, suggesting as before, adhesion of the pleural layers with fibroid changes in the subjacent lung.

Up to December 21st the patient had made no progress, and was still bringing up daily a pint of diffluent, purulent, and somewhat offensive expectoration. Microscopic examinations yielded no trace of tubercle-bacilli; and it may here be remarked that although the sputa were many times examined by Dr. Kidd, pathologist to the hospital, Mr. Downes, the clinical assistant in charge of the case (to whom I am indebted for much valuable help), and myself, no tubercle-bacilli were ever found. The temperature continued to range high, the patient sweated a good deal, and was evidently slowly losing ground. The physical signs remained as last described, except that the axillary area of friction was now completely dull to percussion, and the breath-sounds over it partook of the same cavernous character as those audible behind. Dr. Douglas Powell

and Dr. Kidd now saw the case in consultation, and careful exploratory punctures were again made over the areas of more intense cavernous respiration in the mid-axilla and close to the right nipple, but no fluid was withdrawn. As the evidence pointed so strongly to the cavity which yielded the copious sputa being within the lung base, and the condition of the patient indicated the necessity of operative relief, it was resolved to open the chest-wall posteriorly, and to endeavour to reach and freely drain the cavity through the wound.

Operation by Mr. Marshall.—On December 23rd the patient was placed under ether, and turned on his left side as far as could be done without embarrassment to his breathing. The posterior dull area yielding the cavernous breath-sounds and pectoriloquy, having been most carefully marked out, it was decided to open the thorax at the centre of that space. This point corresponded to the tenth intercostal space just anterior to a perpendicular let fall from the angle of the scapula (see Fig 3); and there Mr.

Physical signs at date of operation.

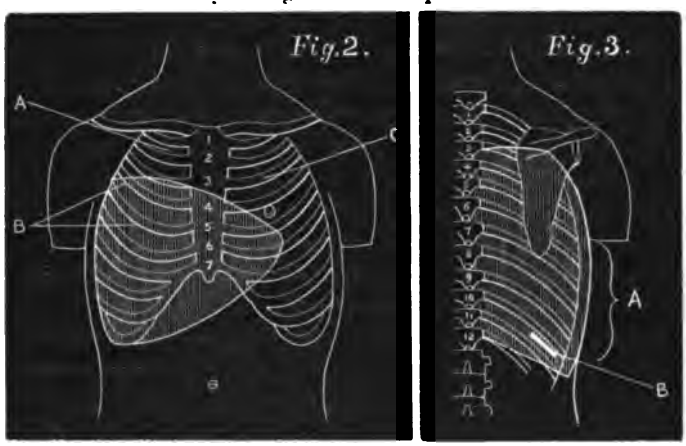


FIG. 2.—A. Weak breath-sounds; no râles. B. Cavernous breath-sounds; pectoriloquy. C. Hyper-resonance; loud breath-sounds.

FIG. 3.—A. Increased vocal fremitus, cavernous breathing and pectoriloquy, gurgling, and crepitant râles. B. Operation incision.

Marshall made a horizontal incision, two inches in length, along the centre of the intercostal space, cautiously deepening it until the pleura was recognised. It was then found that the costal and visceral layers were firmly adherent, as had been anticipated; and after they had been incised the lung-tissue was laid open by a shallow nick or two of the knife, to save bleeding, and a trocar with which a director was combined (an instrument specially designed for such operations by Dr. Hicks, late resident medical officer to the hospital) was pushed into the lung to the depth of two inches. Dark blood flowed, but no pus. A pair of long-pointed dressing-forceps was passed along the groove of the director, expanded, and withdrawn, but no evidence appeared of the cavity having been reached. The trocar was then pressed onwards about two inches deeper, and seemed to enter a cavity for, though no pus flowed, air began to be freely passed out at the wound in short, gasping puffs, and was observed to be distinctly fœtid. A long probe was introduced along the director, and it could be distinctly felt that a cavity had been entered, apparently of no great size, but with an opening at its upper extremity, along which the probe passed with the greatest ease a considerable distance towards the root of the lung. This was clearly a bronchus opening into the cavity, and it was hoped that the object of the operation had been successfully accomplished. A strip of gauze moistened with carbolic oil was packed into the cavity, and a gauze dressing applied. As the patient had spat blood during the operation, he was placed on a draught containing the tincture of digitalis with sulphuric acid, and only cold fluid food administered. No further loss of blood occurred.

On the following afternoon the plug of gauze was withdrawn from the cavity in the lung, and an india-rubber drainage-tube of about half an inch in diameter introduced. No hæmorrhage occurred. The tube was easily kept in place by the pressure of a gauze dressing applied over it, and it was soon found that the wound was draining very freely indeed; the fluid passed out, being thin, purulent,

offensive, and only slightly blood-stained. So profusely did the drainage continue that it became necessary to renew the dressings every few hours, oakum being used with the gauze to obviate the fœtor. The expectoration all but ceased, and the patient professed himself much relieved. Physical examination revealed no notable change in the signs: the position of the cardiac apex-beat was unaffected by the operation.

For the next four days the cavity in the lung was cautiously washed out with Condy's fluid and water, but as this always caused a severe fit of coughing, and the wound was draining very freely, it was discontinued. The whole of the symptoms rapidly improved. The temperature fell to nearly a normal range, appetite returned, the cough was much moderated, and the expectoration was greatly diminished in quantity.

On the 23rd of January the patient was found in the morning much altered for the worse. He had lapsed into a drowsy incoherent state, and about 11 a.m. was suddenly seized with a convulsive attack. He became livid, breathed very hurriedly, coughed continuously and distressingly, seemed unable to expectorate, and presently passed into a comatose condition. On examining his chest loud rhonchus was heard over the sound lung, and it was feared that a quantity of secretion might have found its way thither and occasioned these apparently asphyxial symptoms. This supposition was strengthened by the fact that the drainage-tube was found to have become displaced, and the drainage stopped. The tube was immediately reinstated and the symptoms then subsided. An hour later a fresh paroxysm occurred of the same kind as the former, but this passed off in a short time and the patient had a good night. For the next eight days the case presented no striking change, but no improvement was observed. Severe headache then set in, and in twenty-four hours the patient fell into a comatose condition, cough and expectoration ceased, and he gradually sank and died forty-eight hours later. The cavity drained freely to the last, the wound looked healthy,

but it was noticed that the upper surface of the rib below the incision had become denuded of its periosteum for the whole length of the wound.

The post-mortem examination was made twenty-four hours after death by Dr. Percy Kidd, pathologist to the hospital, and the following particulars are compiled from his notes. A layer of pus was found at the base of the brain. There was a little pus in the lateral ventricles, and their walls were greatly softened and discoloured. In the left temporo-sphenoidal lobe an abscess as large as an egg-plum was discovered, communicating with the posterior horn of the adjacent ventricle; and in the frontal lobe of the same side there was an abscess about as large as a filbert. No miliary tubercles were seen anywhere. On opening the chest, the right lung was found universally adherent except at the right upper sternal border, the adhesions being very firm, and at the posterior and lateral borders of cartilaginous hardness. The margin was firmly adherent to the pericardium. The lung was much contracted, and the overlying pleura enormously thickened and of a fleshy consistency, the thickening being marked even in those processes which dipped down between the lobes. The lung contained a number of multilocular cavities of different sizes, one or two as large as a pigeon's egg, in both upper and lower lobes, which had thin but distinct walls of a dirty-reddish colour and were uninterruptedly continuous with dilated bronchial tubes. These cavities contained whitish, mortary, exceedingly fœtid matter, which was not adherent to the walls, and could be scooped out without any difficulty. No true caseation was observed anywhere, nor any miliary tubercles. The external wound corresponded to a spot about two inches below the bifurcation of the trachea, and communicated with one of the largest of the cavities. The left lung was œdematous and rather congested, its bronchi reddened throughout and filled with frothy secretion. It had a few pleuro-pericardial adhesions. There were no other traces of disease; the other organs were healthy.

Remarks.—It will be seen from the post-mortem report

that the case proved to be one of saccular bronchiectasis limited to one lung, although it wore a different clinical aspect, especially in the earlier part of its history. It is, of course, admitted that the diagnosis of bronchial dilatation was possible from the first, but in several respects the signs varied from those usually characteristic of that affection, and pointed to the possibility of suppuration in the lung itself, or in some part of the pleural sac, with a discharge of the pus through the bronchi. The diagnosis may be briefly passed under review in connection with the three following points:

1. The absence of any history of pulmonary disease prior to the fatal illness.

2. The altered position of the cardiac apex beat.

3. The total absence of vocal fremitus, breath-sounds, and râles, from the dull posterior base of the chest, which was found on the patient's admission to the hospital, persisted for a fortnight longer, and then gave place to noisy cavernous respiration with pectoriloquy and fine crepitation, in a word, to signs signifying excavation of the affected area.

These points will be discussed in succession:

1. Considering the extent of disease found in the lung, the number of the cavities, and size of some of them, it is difficult to resist the conviction that these morbid changes must have occupied a much longer time than the six months' history given by the patient on his admission. It will be remembered that he stated he had never suffered from any pulmonary symptoms before his illness commenced; and therefore, unless this account of himself be totally disregarded, chronic pulmonary catarrh must be shut out of the category of causes which led to the fibrotic changes in the lung. Neither does the history present any other indication suggesting slowly progressive disease. It suggests rather a recent and acute origin; and this point was considered of importance in the formation of the original diagnosis of suppuration within the lung. But upon a review of the whole case it appears most likely that chronic pneumonia, originating from causes that are not obvious and at

a date anterior to the supervention of the symptoms that drove the patient to seek medical relief, must be regarded as the true explanation of the origin of the disease.

2. Doubtless in all cases of fluid accumulations in the pleura, displacement of the cardiac apex is one of the most important diagnostic signs. Again, when the right lung is the subject of chronic fibrosis, it is certain that, sooner or later the resulting contraction will draw over the heart and transfer its apex-impulse to some point at the right of its normal site. In this case the signs were apparently contradictory of the facts: there was no effusion, and there was advanced fibrotic contraction of the right lung, and yet the cardiac apex beat in the fifth intercostal space at a spot a little external to the nipple line.[1] The explanation is difficult, and can only be hypothetically attempted. It may be supposed, and is not improbable, that in the early stage of the disease an effusion took place into the right pleura, displacing the heart leftwards, and originating the pleural and pleuro-pericardial adhesions that were found. Can any force have been in action capable of retaining the heart in its dislocated position after the subsidence of the effusion? If the dynamics which normally control the relative positions of the intrathoracic organs be considered, it will be seen that some explanation is possible.

It has been pointed out and experimentally demonstrated by Dr. Douglas Powell, that "the moment equilibrium between the two lungs is destroyed by the relaxation of the elasticity of one of them, it is restored by the partial contraction of the opposite lung, and the shifting with it of the mediastinum and heart."[2] This condition is produced whenever an effusion takes place into one pleural sac; the elastic tension of the opposite lung gains an advantage proportionate to the relaxation of its fellow, and this traction draws over the heart to the sound side. Supposing then that an effusion occurred in the case under review, and that the absorption of the fluid was followed by

[1] The left lung was normal and there was no cardiac enlargement.

[2] 'On Consumption, and some Diseases of the Lungs and Pleura,' p. 283.

pleural adhesions and rapidly progressing fibrotic changes, tending to impair the normal elastic tension of the right lung, the traction of the left lung would continue acting, and retain the cardiac apex in its position of leftwards displacement. This explanation may not be correct, and is at best hypothetical; but as the importance of cardiac displacement as a physical sign cannot be overrated, anything tending to throw light upon the question is of interest.

3. The total absence of respiratory sounds and vocal fremitus over the posterior dull area, which existed during the first fortnight that the case was under observation in the hospital, is somewhat remarkable when the fact of the number and size of the cavities in the lung and their free communication with the bronchi is considered; and especially in comparison with the intense cavernous sounds heard later on. It can only be explained on the supposition that the cavities were at first loaded with the puriform secretions, and that afterwards the pent-up discharge found a freer vent.

In raising the question of the advisability of attempting paracentesis of basic lung cavities, as illustrated by this case, the first point to be considered is the possibility of estimating justly the risks of operation. Were the cerebral abscesses (obviously pyæmic in their nature) due to septic absorption in the operation-wound, or at some other part of the pulmonary tissue? Now, the pus that flowed from the wound was thin and but very slightly fœtid, while the retained contents of the sacculations of the bronchi were horribly offensive. Moreover, it is clear that the fœtid sputa drained backwards, so to speak, down the bronchi of the sound lung, as was evidenced by the congested and irritated condition of the mucous membranes lining those tubes; and if so, there must have been a continual possibility of infective absorption taking place there. A parallel case has been placed on record by Dr. C. Theodore Williams, in vol. vi of the 'Proceedings of the Medical Society,' in which a patient, suffering from bronchiectasis

with profuse fœtid secretion, was subjected to paracentesis of the lung with great relief to all the symptoms, but nineteen days later he became comatose and died soon after with an abscess in his cerebrum. In this case, also, it might be held that the wound was the seat of the septic absorption, but this is by no means certain. An instructive case has recently been observed at the Brompton Hospital, in which a patient suffering from bronchiectasis in the upper lobe of the right lung, suddenly developed cerebral symptoms and sank. The autopsy revealed an abscess in the left hemisphere of the brain. Here, at least, there was no question of septic absorption from a wound; and in the others it is quite possible that the absorption took place, as in this case, at some point of the pulmonary tissue.

The danger of absorption of the retained secretions in such cases is doubtless great, especially by regurgitation into the sound lung; and it is a grave question whether the operation of paracentesis ought not on this account to be more frequently performed than it is. Abscesses of the lung, pneumonic, embolic, or pyæmic, and the pus-secreting cavities of bronchiectasis in which the puriform secretions are always more or less retained, must rank under the general rule that, in whatever part of the body decomposing matter is confined, and when its presence is evidenced by constitutional disturbance, and a standing risk to life involved by its retention, it must, if at all practicable, be found and evacuated. Probably a larger experience of such operations would prove that their risks were comparatively small and their benefits great.

It is suggested that a concurrence of the following conditions should, ordinarily, determine surgical interference, although, no doubt, there will occasionally be cases in which operative treatment would be justifiable even under other conditions:

1. The diagnosis of the case being distinct, so that the physician is assured that he is dealing with a case of abscess, or a suppurating cavity, *apart from phthisis.*

2. The patient's general state being such as not to forbid the operation.

3. The pleura being adherent over the site selected for paracentesis. If it can be known that such is the case, this would be a distinct encouragement to the performance of the operation; but the contrary ought not to be allowed to be a prohibition. In such a case it would probably be better to prelude the paracentesis by incising the chest-wall and stitching the lung to the wound, so as to induce inflammatory adhesion, and to defer the puncture of the lung until such union was complete, than to resign the operation altogether on the ground of the non-adhesion of the pleural layers. The dangers of pneumothorax are practically not so great as to be prohibitory. The incision of the pleura in such cases ought to be done under strict antiseptic precautions.

The literature of this subject (apart from the question of empyema, irregular cases of which, where the pus has been discharged through the lung, may have closely simulated pulmonary suppurations) is meagre. Dr. Douglas Powell and Mr. Lyell, in an exhaustive paper on this subject printed in vol. lxiii of the 'Transactions' of this Society, have pointed out several reports of cases extant prior to that date. The case described by Dr. C. T. Williams, already cited, is of especial interest, from its similarity to that which forms the subject of this paper. Reference may also be made to a singular case, and one of unusual importance, of "gangrene of the lung treated by incision and drainage" recently reported in the 'Transactions' of the Northumberland and Durham Medical Society for 1883, by Dr. Drinkwater, of Sunderland, in which he opened a gangrenous abscess supposed to be of embolic origin, which formed at the apex of the lung, the pus burrowing and pointing under the pectoral muscles. At the bottom of the wound an opening could be felt in the subjacent intercostal space, and this was enlarged, when the lung underneath was found pulpy and partially solidified. The pleura was adherent. A piece of the third rib having been excised, the abscess

cavity in the lung was reached and cleared out. The patient rapidly and completely recovered. Although this case was not one of basic cavity, nor treated by paracentesis in the way suggested by this paper, it is very pertinent to the present inquiry, because it enforces the principle here urged, namely, that suppurating pulmonary cavities (apart from phthisis) should be opened and drained *under the conditions specified*, with as little hesitation and as good hopes of success as collections of pus in other viscera.

THREE FORMS OF SPINAL DEFORMITY.

BY

W. ARBUTHNOT LANE, M.S., F.R.C.S.,

DEMONSTRATOR OF ANATOMY TO GUY'S HOSPITAL, AND ASSISTANT SURGEON TO THE HOSPITAL FOR SICK CHILDREN, GREAT ORMOND STREET.

(Received February 13th, 1884.—Read June 6th, 1884.)

Looking through works of surgeons who have paid special attention to the subject of spinal curvature, I find in them no definite reference to the forms of spinal deformity produced by the occupation of the individual.

In a vague way, cases of curvature commencing in the dorsal region and followed by compensatory curves are referred to, as a form of ordinary lateral curvature, but no pathological information is given. I may quote the most recent English work ('On Curvatures and Disease of the Spine') by Brodhurst. In it he writes, "Again, other causes of spinal curvature exist, namely, such as increase or diminish the power of a single limb. Thus the power may be increased by extraordinary use, as through carrying a burden on one arm, or as occurs in certain trades, where one arm is much more employed than the other. And it is found that nurses, needlewomen, tailors, shoemakers, compositors, and some others are unusually liable to spinal curvature through the inordinate use of one arm."[1] As these

[1] 3rd edit., p. 15.

deformities are so common, I must say I am much surprised at not finding any described specimens in our museums, though I hope to show that some are present under other names.

Their frequency in the dissecting room is easily accounted for, as the supply of male subjects is drawn from a class who have to work hard for their living, and who have usually had some heavy manual labour to perform.

In this paper I shall describe three distinct forms, of each of which I have several pathological specimens, and I will take them in their order of frequency, the first being the most common, and occurring in men whose occupation is to carry heavy and bulky burdens on their right shoulder, as, for example, draymen, who carry barrels of beer in that way.

Let a strong man be made to bear on his right shoulder a heavy body as a barrel of beer, and you find he is no longer able to remain in the erect position. His head and neck are thrown forwards and to the left, partly that his head shall be out of the way of the object, but chiefly that its weight shall be transmitted more directly to the spinal column.

The upper dorsal spine will be found to share in the forward flexion of the cervical vertebræ.

The spines of the vertebral column will now be seen to form an arc with its convexity to the right side, the most convex portion corresponding to the position of the sixth dorsal vertebra. The curve is but slightly marked in the lumbar region, being chiefly limited to the dorsal and lower cervical spine. It is a long single curve, and there is no indication of any compensatory curve, which is only what one would expect. The chest wall is seen to be altered in shape. The left half of the chest is thrown more forwards and the right half backwards, so that the right chest is more prominent behind than the left, especially below. The ribs here are also found to be separated by a larger interval from one another than on the left side. This is owing to the rotation of the vertebræ which

accompanies the lateral flexion, and is an attempt on the part of nature to form a larger supporting basis which will transmit the weight to the spine partly through muscles attached to the ribs and partly through the leverage they possess on the bodies and transverse processes of the vertebræ.

The right upper ribs cannot be observed owing to the presence of the weight. One can, however, feel and see that they must support the greater part of the weight, and must transmit it to the spine. The rest of the weight is sustained by the muscles which support the scapula or arm, and partly by the articulations of the clavicle and sternum and costal cartilage, this last being an indirect means of support.

Now, suppose we go a step further than we can see in our subject, and follow in our imagination the changes that ought to take place in the osseous and ligamentous tissues of a man whose occupation it is to carry these heavy weights frequently, and for some time. Owing to the frequent lateral flexion of the spine, the intervertebral substances, where they correspond to the concavity of the curve, become thinned, perhaps develop a synovial membrane (Gelenkhöhle of Luschka), or become completely destroyed, and the bones may come together and fuse, and throw out from their margin large osteoplastic growth.

Also the anterior portion of the intervertebral substances between the upper dorsal vertebræ will become thinned owing to the increased antero-posterior flexion of this portion of the spine, and lipping of the vertebræ may also take place.

This lipping and thickening of the vertebral margins is probably an attempt to increase the breadth of the column where it is most needed, which is of course at the concavity of a curve.

The upper ribs on the right side are pressed downwards and forwards by the weight which rests upon them, and they by their immense leverage on the transverse processes of the upper four or five dorsal vertebræ will tend to

rotate their bodies to the opposite side, namely, to the left, and will render this portion of the dorsal curve which was at first convex to the right, now almost a straight line, and then later, convex to the opposite side. This secondary curve will extend, however, slightly if at all to the left of a line joining the upper cervical vertebræ and lower lumbar.

Accompanying this rotation of the upper dorsal vertebræ you get the upper ribs on the left side raised somewhat, the intervals between them being increased in size, and their angles project more backwards. The opposite condition has taken place in the upper right ribs. The single convex line formed by the spines of the upper dorsal vertebræ is unaffected by this rotation of their bodies. If any change takes place it will be in the direction of an increase of the convexity of the curve to the right. In the case of the leverage of the rib, the fulcrum corresponds to the articulation of its head with the body and intervertebral substance, and the resistance or weight to the ligamentous attachment of the tubercle and neck to the transverse processes. So that in examining a living subject, one would not be able to recognise the presence of the secondary (not compensatory) curve by the position of the spines, as only one curve would be visible.

Owing to the formation of the upper curve, you would expect an increased destruction of the left half of the intervertebral substances and vertebræ corresponding to the concavity of the primary curve, and this will be most marked about the fifth and sixth dorsal vertebræ and a similar progressive destruction corresponding to the concavity of the secondary or upper dorsal curve. The middle ribs on the left side will be still more approximated.

Now, to make up for this damage to the upper part of the dorsal curve, that the weight may be transmitted as much as possible to the spine, an increase in the primary curve and increased rotation of the bodies of the lower six or seven dorsal vertebræ must take place, resulting in

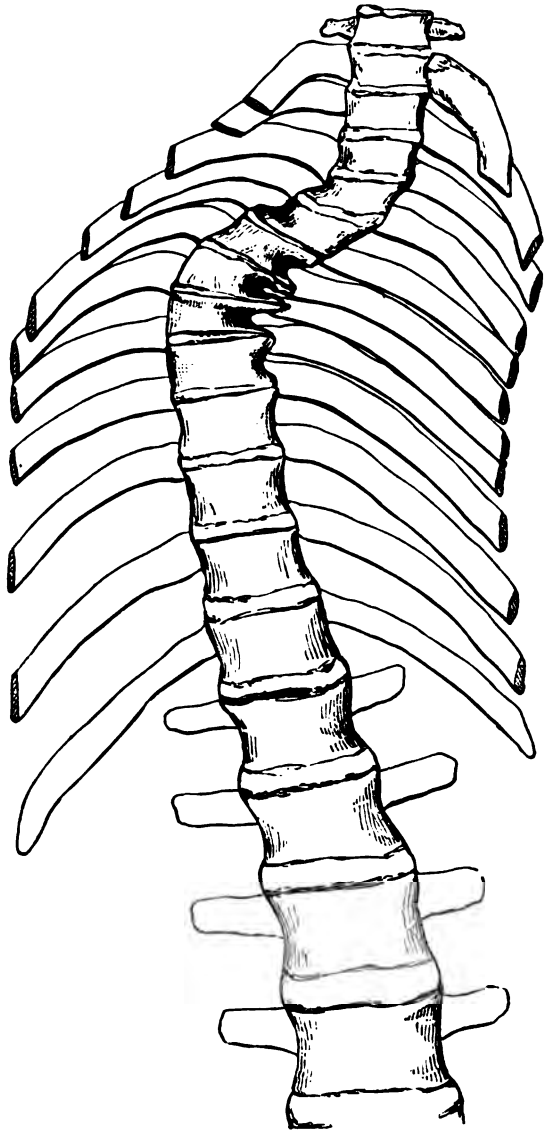


Fig. 1.

a greater destruction of intervertebral substance and vertebræ at the concavity of curve, most marked at the union of the two curves, with changes in the lower ribs as before described. This is by no means as simple a process as is the change of shape of the chest which one sees in the ordinary lateral curvature which develops in young adult life.

Fig. 1 shows exactly the changes we have arrived at synthetically. I have chosen this specimen as it is the best marked of the five specimens which I possess of this form of deformity. It was obtained from the body of a great, powerfully-built man, who had been a drayman. He was about forty-five years of age, and had all his teeth. His muscles were in excellent condition, and his bones dense and strong. Death had been due to heart disease. Till the chest was opened, while the body was lying on its back, there was no indication of the condition of the spine. I would particularly call attention to the fact that in this condition there is no compensatory curve or rotation in the lumbar region, the articular processes of the lower lumbar vertebræ being in a transverse plane, while the upper lumbar vertebræ share in the primary curve. Except for the secondary curve in the upper dorsal region there is no rotation of the bodies of the vertebræ to the left. I mention this as in the forms of lateral curvature described, most authors will not allow that a rotation to one side can exist without a compensatory curve to the opposite side. One can easily see that the formation of a compensatory curve in the lumbar region to the opposite side would be detrimental. What has taken place is simply that the spine has been bent in such a way so that with the ribs it forms a sort of shelf on which the weight rests, and by which the pressure is transmitted equally to the legs. By placing this specimen in the normal position, and by applying pressure on the upper ribs on the right side, the purpose of the curvature and the mode of its formation can be seen very plainly. A practical point I would call attention to, as exemplified by the upper

dorsal curve, or secondary curve as I have called it in this paper, is the immense power that can be exercised by the ribs in rotating the vertebræ. This explains the benefit sometimes observed from pressure well applied to the side of the chest, and also indicates that it should be made to act in a direction obliquely downwards and inwards rather than inwards or upwards and inwards. Out of thirty-five male subjects dissected during the winter session, I found five very well-marked examples of this curvature. They were almost exactly identical in character, but varied somewhat in degree.

The second condition is one which is present in men who carry heavy bulky weights on their backs, as coal-heavers, &c. If one watches a coalheaver at work, one sees that, when carrying a load, his body is bent forwards. The weight is seen to rest on the back of his head, neck, and trunk, being retained in position by both hands.

Let us attempt to arrive at the changes one would expect to find in the bones and ligaments of a man who had pursued this occupation for many years. If dying before his tissues had commenced to degenerate, the osseous tissue of the vertebræ will be found to be very dense. This is most marked in the cervical and upper dorsal region on account of the small size of the bodies of the vertebræ in this region. The weight, being a rigid one, diminishes the natural convexity of the upper part of the dorsal spine, partly by direct pressure, and partly indirectly by pressure on the ribs, so rendering the upper two thirds of the spine nearly straight.

The force then exerted on the flexed lumbar spine by the chest with its load, and by the load itself pressing directly on the lumbar spine may be resolved into two component forces, the one acting at right angles to the upper and lower surfaces of the bodies of the lower dorsal and lumbar vertebræ in a direction parallel, and the other at right angles to their opposing surfaces.

These will cause, first, an increase in the lumbar curve.

Where the straightened dorsal spine joins with the convex curve of the lower dorsal and upper lumbar vertebræ there will be destruction of the bodies and intervertebral substances of the tenth, eleventh, and twelfth dorsal and first and second lumbar vertebræ, especially in front.

The spine at this point may not deviate laterally, but this occasionally happens owing to the unequal lateral destruction of the bodies of the vertebræ, due probably to some inequality in the support given by either arm.

The bodies of the third, fourth, and fifth lumbar vertebræ with the intervening fibro-cartilages, as they form part of the convexity of an antero-posterior curve, will be but little diminished in depth in front.

Owing to the sudden transition from the convex lumbar curve to the concave sacral one, the force tending to displace the lumbar vertebræ forwards and downwards acts at this point at an immense advantage.

This will cause the fifth lumbar vertebra to become displaced forwards more or less, so that it projects beyond the sacrum. In the interarticular fibro-cartilage between it and the sacrum there is formed a large articular cavity, and from the front of the upper piece of the sacrum is formed a projecting shelf of bone which helps to support the displaced vertebra. The articular cavity is the result of the considerable restitution after the weight pressing on it has been removed.

The laminæ of the fifth lumbar are closely approximated to the posterior margin of the body of the first sacral vertebræ diminishing considerably the calibre of the spinal canal, and interfering in the same way with the size of the intervertebral foramina between the sacrum and fifth lumbar. The form and position of the articular processes and laminæ are naturally much altered by the prolonged pressure.

I do not find the least indication of any congenital deficiency of the laminæ or articular processes, nor any sign of any previous injury or fracture. I refer to this particularly, as Neugebauer, in his article on spondylolisthesis,

takes rather an opposite view.[1] I see no reason to suppose that in the production of spondylolisthesis in this form of spinal deformity it is necessary to suppose the existence of anything beyond great pressure acting at a great mechanical advantage upon the articulation. Criticising Neugebauer, I think that he has erred in regarding the displacement of the fifth lumbar as *something quite peculiar and local and not as part of* a general change in the spinal column. I do not think that in any of the examples he describes he has given us any account of the condition of the rest of the column.

The pressure of the weight is also transmitted to the neck partly directly and partly indirectly through tension exerted on the head by the tarpaulin cap, or other means of support used by this class of labourer. This renders the cervical spine more convex forwards, and causes pressure changes in the lower cervical vertebræ and fibro-cartilages.

Fig. 2 is a drawing of the spine of a middle-aged man. He was powerfully built. I was unable to discover his occupation. The deformity it exhibits corresponds to those I have just described. The intervertebral substances between the fifth, sixth, and seventh cervical vertebræ have become thinned, and have developed articular cavities in their centres. The bodies of these vertebræ are diminished in depth, and their lipped margins project forwards, and backwards into the spinal canal.

The odontoid process presents marked signs of the pressure that has been exerted on it by the anterior arch of the atlas. Its facet is much increased in size, and from its upper margin there is an outgrowth of bone to strengthen the articulation, and increasing the height of the process. Owing to the removal of bone from its anterior surface, the antero-posterior diameter of the process is diminished. The arch of the atlas presents

[1] 'Zur Entwicklungsgeschichte des spondylolisthetischen Beckens und sei ner Diagnose,' von Franz Ludwig Neugebauer.

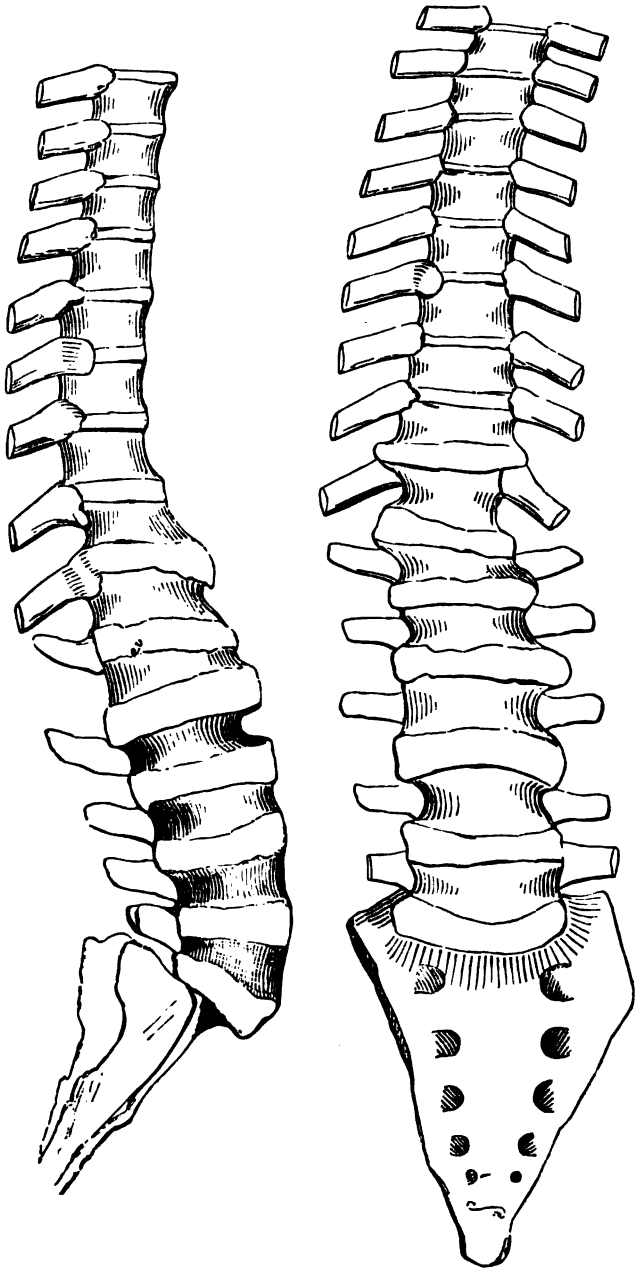


FIG. 2.

corresponding changes. Two out of the thirty-five male subjects possessed spinal columns which presented the above conditions.

While watching labourers carrying heavy weights on their heads, I have often speculated as to what changes one would expect to find in their bodies as the result of this form of pressure. These men, when bearing a load (which is often a very heavy one), allow the head to sink somewhat downwards towards the shoulders and do not keep the cervical spine extended.

They keep the load in position by one or both hands. I concluded that the cervical spine would present a well-marked lateral curve, with the accompanying destruction of cartilage and bone on the concave side. Below this large cervical curve one or two compensatory lateral curves occupying the dorsal region, the extent of deflection of the curves from the median line diminishing as they descend.

I was some time before I discovered a subject in which the spine presented changes similar to those I expected to find. Fig. 3 is a drawing of the first one, and I will now describe it in detail.

The articulations between the atlas and occiput and atlas and axis present but a slight deviation from the normal condition, and this was limited to slight asymmetry of the articular facets. The axis and third cervical vertebræ are fused together, the fusing being more complete on the left side. This causes a slight lateral displacement of the atlas. Below this there is a large lateral curve with its convexity to the right, the body of the sixth cervical forming the centre of the arc. The vertebræ are here very dense. The left halves of the bodies are much diminished in depth, being rendered somewhat wedge-shaped. The fibro-cartilages, especially in the concavity of the curve, are much destroyed, more so between the upper than the lower cervical vertebræ. In fact, as the fibro-cartilages ascend they diminish in thickness, being almost completely absent between the third and fourth

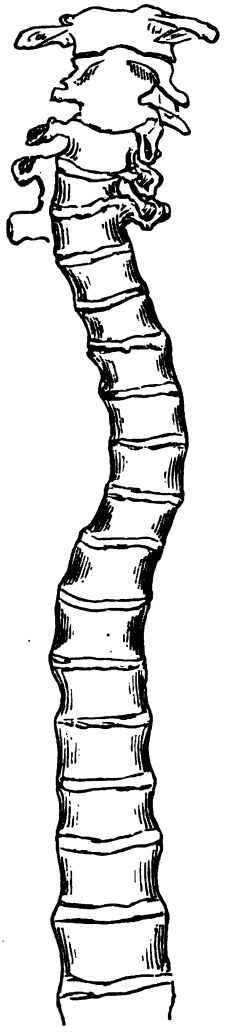


FIG. 3.

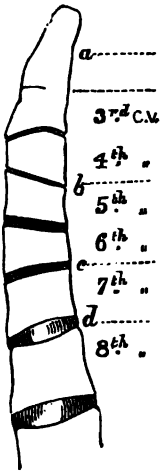


FIG. 4.

vertebræ, so that one sees a regular gradation from normal fibro-cartilage below, to osseous union of the vertebræ above. Fig. 4 is a vertical section through the 2nd, 3rd, 4th, 5th, 6th, 7th, and 8th cervical vertebræ, and shows these changes. The articular processes are also altered in form by the pressure. Below the cervical curve there is a compensatory curve with its convexity to the left, and again below this there is less marked curve to the right. Later I found another example of this deformity among the thirty-five male subjects before referred to.

In the museum of Guy's Hospital I find many specimens of fusion of the atlas and axis, but without the remainder of the spine. As in the case I have just described, the bones are unequally fused so that union is more complete and advanced on the left side than on the other. This obliquity of union alone indicates that there must have been a compensatory curve lower down. I have no doubt that this was due to the same cause that produced similar osseous ankylosis in my case, and which I believe is force transmitted through the occiput.

I think that there will be found many other distinct forms of deformity besides those I have described, also modifications of those three forms due to individual differences in the method of carrying and supporting the loads.

There is another condition to which I would like to have paid more attention, and that is the fusion of the occiput and atlas, and sometimes also the axis as well. There are many examples of these in our museum. Unfortunately this portion only of the column is present, so that one cannot venture any definite hypothesis as to the particular means by which force is transmitted in this case, as I believe it must be the cause here also of the fusion.

I have for a long time looked in the dissecting room for a spine showing this abnormality, but have as yet failed to find one.

I also believe that many museum specimens put up as examples of rheumatic arthritis, osteophytic growth, and

congenital deformities of the bodies of the vertebræ are merely changes produced as a result of pressure.

That there is a greater tendency to the formation of compensatory bony outgrowth and lipping of the vertebræ if the man is also affected by rheumatic arthritis is probable, but I have not yet been quite able to satisfy myself on this point. At the same time, I believe that the irritant poison in rheumatoid arthritis can of itself produce destruction of cartilage and osseous ankylosis in articulations kept perfectly at rest, as in those of the tarsus. This I have referred to elsewhere (Mollities ossium;—Charcot's disease;—Rheumatoid arthritis, 'Trans. Path. Soc.,' 1884).

I am sure, however, that many spines are said to present rheumatoid change when it is simply the result of a physiological and healthy process.

That changes from the normal curvatures of the spine are by no means uncommon in the bodies of hardworked labourers is shown by the fact that in thirty-five male subjects dissected consecutively, I found five well-marked examples of the first class of deformity, two of the second class, two of the third, and besides these four species presenting changes which did not belong to either of these classes. In all there were thirteen deformed spinal columns in thirty-five subjects.

ON A PARTICULAR FORM OF AMNESIA.

LOSS OF NOUNS.

BY

W. H. BROADBENT, M.D.,

PHYSICIAN TO, AND LECTURER ON MEDICINE AT, ST. MARY'S HOSPITAL; CONSULTING PHYSICIAN TO THE LONDON FEVER HOSPITAL.

Received February 25th—Read June 10th, 1884.

On two previous occasions I have brought before the Society cases of affection of speech with post-mortem examinations, and have endeavoured by their means to elucidate the mechanism of speech and thought.*

The opportunity has just been afforded me of examining the brain of a patient who presented a form of defect of language the importance of which has scarcely been adequately appreciated, the defect consisting in the entire loss of the power of uttering nouns substantive, whether for employment in speech or by way of imitation.

The patient, a gentleman at that time æt. 72, was first seen in consultation with Mr. Meehan, of Regent's Park, on September 17th, 1878. On the 12th he had been seized with faintness while sitting in a hot greenhouse in the Botanical Gardens and had for a time entirely lost his

[1] "On the Cerebral Mechanism of Speech and Thought," 'Med.-Chir. Trans.,' lv, p. 145; "On a Case of Amnesia," ib., lxi, p. 147.

speech. This he partially recovered, while slight hemiplegia of the right side had gradually come on. At the first consultation the precise character and extent of the speech defect could not be defined; there was conspicuous paralysis of the right side of the face, of the kind seen in hemiplegia, a moderate degree of paralysis of the upper extremity, slight paralysis of the lower extremity; with this, marked hemianæsthesia and loss of the sole reflex. The patient was suffering from severe pain in the right temple.

He speedily recovered so far as to be able to go about and to cut up his food with the right hand, and the affection of speech had in November reached the point at which it remained for more than five years, during which time he was continuously under the care of Mr. Meehan, and was carefully watched by intelligent relatives and by a nurse of remarkable power of observation and accuracy of description, whose attention was specially directed to the peculiarity of the speech. I saw him at intervals, and in his final illness, which was an attack of congestion of the lungs.

During the whole of this time he was scarcely ever known to utter a noun substantive, and if he did, it was, so to speak, inadvertently and erroneously. Other words he said unhesitatingly, and he would employ fairly long phrases, speaking them smoothly and naturally so long as a noun did not come in his way. The following are examples: "I am very glad to see you," "I am very much better to-day, thank you," "Oh, yes, very well indeed," "I cannot remember it at all." When he wished for anything he would say "I want the one," "Please give me the one." And the nurse would, by guesses, find out what it was, when he would say, "Yes, that's it, thank you." Asked his age he replied, "I shall be seventy-three on the 3rd—4th"—stopping puzzled and baffled—he meant on the 24th of December. He was always brought to a standstill by a noun, and was at first greatly distressed and confused by his efforts to remember

the word. He was unable to name an object at sight—the hand, or a pen, or the sheet; and not only this, but he could not repeat after me the above words, though he tried hard. Once when endeavouring to say hand he said something like leg. When told to say one, two, three, during the examination of his chest, he did so promptly.

He clearly understood all that was said to him and took a great interest in all that went on in the household. He read his newspaper and often pointed out paragraphs relating to people and places that he knew, showing that he understood it. His memory was said to be remarkably good and accurate. He could sign his name, but could not answer a question in writing.

He was cheerful but emotional, often crying when I first spoke to him and in saying good-bye.

The paralysis entirely disappeared, but the nurse stated that sensation did not appear to be so good on the right as on the left side. The patient did not notice when the right foot slipped from the footstool as it was liable to do.

Such was the condition. The patient died on December 22nd, and the examination was made on the 24th, about forty-eight hours after death.

The cranium was very thick and dense, and the brain apparently somewhat shrunken, but not more on one side than on the other; there was no fluid in the membranes over the hemispheres and not much at the base. Inspected before its removal, nothing abnormal was discovered about the brain; in the act of removing it the membranes and nerves were observed to be very tender and lacerable and the arteries moderately affected with atheroma; the brain substance was soft.

The corpus callosum having given way along the median raphé, the detailed examination was begun by completing the division of this commissure and by removal of the velum interpositum and fornix, the latter being torn. The left thalamus was smaller than the right, but neither on its surface nor on section was any focus of morbid change visible. The anterior rounded extremity of the

left corpus striatum had its natural size and appearance, but as it narrowed, two digital depressions were seen, one just in front of the posterior border of the thalamus, the other a little beyond this body ; there was no discoloration, and nothing was apparent on section at these points but a local loss of substance. The most conspicuous change was in the posterior cornu of the ventricle, where a brownish translucent patch, rather larger than a sixpence, occupied the situation of the calcar avis. A band of white fibres, apparently untouched by degeneration, ran from the thalamus along the inner side of the patch to the tip of the occipital lobe. From the translucent area itself all trace of nervous structures had disappeared, leaving only the pia mater lining the calcarine fissure, and this was not apparently vascular. The comparison between the corresponding part of the two posterior cornua was very striking, the right calcar presenting a smooth white prominence over which ramified a vessel of some size. The descending cornu was large, the fibres along its roof soft and apparently wasted, but the hippocampus with its tænia was in a normal condition.

The right hemisphere was normal in all respects.

In the left hemisphere the convolutions of the convexity everywhere presented a natural appearance. The third frontal and adjacent part or foot of the anterior or ascending frontal gyrus were specially examined on all sides, including the surface within the fissure of Sylvius, and found to be normal.

The Sylvian fissure gaped somewhat and the pia mater over it was thickened. When the insula was exposed it was seen to be unduly smooth. Of its six convolutions the two anterior which dip under the posterior margin of the orbital lobule were in their normal state ; so also was the first of the two outer ones which correspond with the end of the third frontal and the foot of the first ascending gyri ; the more posterior of these was reached but not destroyed by degeneration. The two long convolutions which run from the summit of the insula towards the

posterior extremity of the fissure had entirely disappeared, leaving a smooth surface of a pale fawn colour, formed apparently by the external capsule, itself somewhat degenerated but overlying grey substance of the lenticular nucleus which was not appreciably altered. The angular gyrus round the extremity of the Sylvian fissure and the supra-marginal lobule forming its upper margin posteriorly, were undermined by an extensive area of degeneration continuous with that which had destroyed the posterior insular convolutions. The degeneration, however, did not reach the surface of the gyri presenting on the convexity of the hemisphere, or penetrate very deeply into their white axial fibres.

A series of transverse sections revealed nothing more. Parts of the brain and cranial contents not mentioned may be taken as presenting nothing abnormal.

Before commenting on this case I may relate another, that of a lady æt. 75, seen with the late Dr. Frampton in October and November, 1879.

She had been a little out of sorts on the 12th of October, and on the morning of the 13th mumbled and could not be understood. There was some improvement during the day, but after sleep at night she woke in a state of extraordinary excitement. She was out of bed in a moment and round the room more quickly than she had been for years; she was talking rapidly, but no one could make out what she wanted, which made her very angry.

When I saw her on the 20th she was quiet and rational, ladylike and lively. She understood all that was said, and answered many questions that did not require the use of a noun. She asked anxiously and fluently, "Shall I get better of this . . . ?" trying to complete the sentence, but stopped by her inability to find the noun she required. Other phrases which I took down were, "I hope you will be able to do me good . . . to take this away." She was encouraged to tell her own tale, and spoke distinctly and unhesitatingly, but got confused when she came to a noun. She resented all attempts on the part of her maid to assist

her and told her not to speak. She could not name a watch, the curtains, or Dr. Frampton, and could not tell her age. She was moreover quite unable to read, having up to the moment of the attack been a great novel reader. There was no paralysis of any kind.

At a second visit in November she recognised me and received me in a natural and ladylike way. She said, "Oh yes, I remember you quite well," and used other similar phrases. I sat by her bedside for a quarter of an hour, during most of which she was talking, but except that she once said "thing" and once inappropriately "jealousy," and two or three times "the other day" adverbially she did not employ a single noun substantive. On coming to a noun she bungled and employed some unintelligible substitute.

This lady lived some time, and I understood from Dr. Frampton that the speech did not change, but I did not see her again.

In a third case seen with Dr. Marcus Allen at Brighton, and subsequently in London with Mr. J. Ernest Lane, loss of nouns was a stage in disease which advanced to complete loss of speech and to a rapidly fatal termination.

The extraordinary analysis of language made by disease of the brain in cases such as these will strike anyone who gives the subject a moment's thought. A single "part of speech," the noun substantive, is separated from all the others, and while they can be spoken fluently and appropriately, utterance of nouns of all kinds fails entirely; they do not come for the expression of thoughts or wants, they are not suggested by objects presented to the sight, and they cannot be repeated imitatively. This analysis by disease is all the more remarkable, inasmuch as it corresponds with the analysis of language arrived at by logicians and grammarians. They also place nouns substantive or names apart by themselves, a distinction recognised in German by the capital letter with which they are spelt. Nouns or names are the objective material of thought—the subjects of predication—other parts

of speech are merely a framework in which names are set for comparison or predication.

Again from a physiological as well as from a philological point of view nouns or names stand alone. They only are intellectual symbols properly so-called. As I have attempted to set forth in my former communications they represent the final act of intellectual elaboration of the perceptions derived from the different senses. A complete elementary notion of an external object is a combination of all the perceptions, visual, tactile, &c., and the auditory perception associated with this notion in early childhood is the *name* which thenceforward habitually symbolises the object. A noun raises a mental picture which is not the case with any other word. It belongs, in effect, to the sensory side of language of which it is the highest point, while all other parts of speech imply movement of ideas and may be said to be motor.

In order that the bearings of these considerations and of the case as a whole on the mechanism of speech and thought may be explained, it will be necessary to restate briefly some of the conclusions arrived at in my previous papers, and especially to discuss the exact part played by the third left frontal gyrus in articulate speech. This has been admirably expressed by Dr. Hughlings Jackson in the phrase "the way out for words," or it may be otherwise described as the downward starting-point of language; its function is essentially motor, like that of the cortical motor centres in the region of Rolando, and its cortical cells may be compared to the motor cells in the anterior grey cornua of the spinal cord. These cells, constituting the motor nerve nuclei in the cord, are co-ordinated or grouped for the production of orderly movements through the agency of sensory nerve-cells connected with the posterior nerve roots, and the motor cell groups so formed are then ready for employment, and are employed by higher centres. In the same way, then, as the cells of the anterior grey cornua are grouped or educated by sensory cells on the same level, the cortex of

Broca's convolution will be educated for the utterance of words by the auditory perceptive centre, the superior temporo-sphenoidal gyrus, which is on a level with it anatomically as well as physiologically (being, like it, a marginal convolution), and being educated, *i.e.* its cells being properly grouped, will be employed by higher centres which are the seat of the intellectual operations. Again, just as the integrity of the sensory side of the spinal apparatus is necessary to the functional efficiency of the motor cell groups, and damage to the former, as in locomotor ataxy, gives rise to inco-ordination, so with regard to the auditory perceptive centre and the motor word centre.

But, as has already been said, language has its sensory aspect and its sensory cortical apparatus. I have put forward in previous communications, as a tentative hypothesis based upon observed cases, the supposition that each sense having its perceptive centre in the convolutions identified as such by Dr. Ferrier, the perceptions from these several cortical areas converge, by means of tracts of fibres, upon a higher cortical region in which they are combined and elaborated into an idea which is symbolised by a name. This naming centre or idea centre will be the highest centre on the sensory side of the nervous mechanism, and the corresponding motor centre I have supposed to be a propositionising centre in which the first motor process of language and thought, the mental rehearsal of a phrase, takes place.

Taking these assumptions for a moment as granted, it is conceivable, first, that the paths from the visual and auditory perceptive centres to the naming centre may be destroyed. In case of lesion of the communication between the visual and naming centres the sight of an object would no longer call up the name. Such a case I have related in the first of my papers read before this Society, describing the lesion found after death. A patient, otherwise perfectly intelligent and who conversed fluently, was utterly unable to name an object at sight.

Tested scores of times over a period of many months he never once succeeded in naming such familiar things as a pen, chair, table, hand, coat, or any object whatever. Obviously a man in this condition would be unable to read, and such was the case with the patient; what made it more interesting was that he wrote correctly out of his own head or from dictation, but could not read a single word of what he had himself written.

Such cases have been described as examples of "word-blindness," but this term is altogether inadequate and misleading. The word-blindness is only a part of a wider and more significant deficiency. It is not as words that words cannnot be read, but as visual characters or objects which cannot be named.

A corresponding interruption between the auditory perceptive centre and the naming centre will have more striking results. The subject of the lesion will understand nothing that is said to him and he may or may not know what he says to others. Many examples of this condition are on record, and I have narrated such a case, in the paper already referred to, but without post-mortem examination.

The patient was a lady who replied quite inappropriately but in intelligible and connected sentences to questions and observations, and showed that she did not understand what was said to her by failing to do simple things when told.

Again, the naming centre may itself be destroyed, and under the title of a Case of Amnesia I brought before this Society what I believe to have been an example of such a lesion, in which, without any damage to the ascending convolutions of Rolando or to the third left frontal gyrus, but with extensive softening of convolutions behind the motor area in the left hemisphere, a patient was utterly unable to comprehend spoken or written language, and talked incomprehensible gibberish in which not a single word was, as a rule, recognisable; he spoke his inarticulate jargon fluently and with natural emphasis and intona-

tion, and evidently had ideas in his mind which he thought he was expressing.

Returning to the case here related it is supposed to illustrate breach of the communication between the name centre and the propositionising centre. The patient had in his mind ideas and wishes, and rehearsed mentally, and uttered in words, phrases expressive of these ideas and wishes all but the nouns or names. These were not supplied in consequence of the interruption of the tract from the name centre to the propositionising centre.

The lesions found after death are interesting from whatever point of view they may be considered. One reflection suggested is that the exclusive employment of the left hemisphere in the utterance of speech involves its predominant if not exclusive employment in the integration of language in thought. Again, the case illustrates the fact that there may be loss of speech without damage to the third frontal convolution, but shows, at the same time, that the cases in which this occurs are recognisable during life as distinct from aphasia as commonly seen.

Observing upon the lesions *seriatim*, the calcar avis or hippocampus minor is no longer looked upon as the distinguishing peculiarity of the human brain, and it is not associated with language in any theory known to me. Whatever effects the destruction of this part in the left posterior cornu may have, it is not probable that it had any share in the affection of speech in the case under discussion. Nor can the unimportant loss of substance noticed in the thalamus and intraventricular corpus striatum have had any such effects. Clearly the important lesion was the extensive area of degeneration situate in the posterior part of the Sylvian fissure. The structures destroyed would be as follows :

a. The two posterior convolutions and part of the posterior external convolution of the insula with the white fibres which arise in the grey cortex of these convolutions, and pass almost entirely to the adjacent convolutions of

the parietal lobe overhanging the fissure, the supra-marginal lobule and part of the angular gyrus.

b. The grey matter and subjacent white fibres of the convolutions forming the upper wall of the Sylvian fissure behind the posterior ascending convolution of Rolando, viz. of the supra-marginal and angular gyri just mentioned. The degeneration involved the white substance to a considerable depth, undermining the convolutions, but not implicating their entire thickness, and it did not reach anywhere the summit of the gyri seen on the convexity of the hemisphere.

c. The posterior part of the claustrum, together with a mass of fibres underlying the posterior convolutions of the island, and running from behind forwards in the deep angle of the fissure. These fibres converging from various sources round the posterior end of the fissure, the angular, supra- and infra-marginal, parallel, and other gyri, form a large bundle or plane of fibres, which have a longitudinal course along the deep angle of the fissure, and pass to the foot of the two ascending parietal convolutions and to the posterior part of the third frontal. They constitute a kind of longitudinal commissure.

d. The posterior part of the external capsule, which is composed of fibres arising in the grey matter of the extra-ventricular corpus striatum and radiating to the fronto-parietal part of the hemisphere, after many of them have decussated with the fibres of the internal capsule.

The infra-marginal or first temporo-sphenoidal convolution, the seat of the auditory perceptive centre, had escaped damage in a remarkable way, and, on section, the grey matter of the extra-ventricular corpus striatum was seen not to have been reached.

It is to lesion of the longitudinal commissural fibres that I attribute the loss of nouns, as cutting off the naming centre from the motor speech apparatus. Those who have looked upon the island of Reil as the seat of the cell and fibre mechanism of speech may of course claim the case as supporting their view.

There are other remarkable facts observed in connection with loss of speech the significance of which has not yet been fathomed, which I therefore venture to mention briefly. One of these is the retention of the power of saying numbers when no other word can be said except "yes" and "no," and perhaps an interjectional exclamation. A patient, admitted into St. Mary's Hospital for right hemiplegia and loss of speech, diagnosed as due to hæmorrhage, while unable to say anything but "yes" and "no" could count up to twenty, could say at once how many pence there were in a shilling and how many shillings in a pound, and named without hesitation the number of coins shown to him; now and then he would say not only "two" or "five," but "two shillings" or "five shillings," being utterly unable to say "shillings" when told to repeat the word; the numeral dragged with it the noun. He would say "two and six" when shown this amount, but never could be got to say "half a crown."

A man now attending from time to time and who has been under observation for five or six years, can say only "yes" and "no" or sometimes indistinctly what is evidently "No, I can't." He cannot even repeat "no" when told to do so after he has just given that answer, the word being said over and over again for him to imitate. Once or twice after prolonged effort he has given it up in despair with a negative shake of the head and saying "No, I can't." This patient till quite recently could count up to twenty but never beyond, and he sometimes got confused at twelve and thirteen. He has always been able to tell us the number of spokes in a wheel (he is a wheelwright), twelve or fourteen, by counting up to the number and then stopping. He will give the number of coins shown to him, counting inaudibly but visibly. He writes numbers in figures much more promptly than he says them and without preliminary counting, as, for example, the number of fits he has had, the number of weeks since he came to the hospital, the amount of his earnings, &c. He usually gives these answers by

writing the figures on the palm of his hand, and he can count in writing up to thirty, forty, &c. He cannot write "yes" or "no," but he has been taught to write something like "Tring," to which place he has recently removed from town. He can show numbers up to ten on his fingers. Except numbers and the two or three other words mentioned, he cannot say a single word, and if told to repeat the alphabet makes futile attempts and often begins to count.

Such cases as these are not mere curiosities nor are they very great rarities. Other illustrations have come under my observation, and during a visit to St. Mary's Hospital Professor Gairdner described a case corresponding so exactly with the second of those just given that, the patient entering the ward at the time, I was able to say, "Why, here is your patient."

Provision will have to be made for them in any complete scheme of the mechanism of speech and thought. The explanation cannot lie in any distinction between emotional and intellectual expression such as is supposed to account for the utterance of oaths and interjections, and the retention of yes and no when no other words can be spoken. Number is an elementary abstract conception, and the idea of number is arrived at by a different process from that by which the formation of ideas of concrete objects is accomplished. In numerical computations our thoughts are turned inwards, so to speak, while in matters of form, &c., they are projected outwards. It is probably in relation with this difference that numbers can be spoken and written when no other words can. There is every reason to believe that in the cases cited the third left frontal gyrus is destroyed, and it is inconceivable that a small portion, concerned only with figures, should be spared.

As has been stated in previous communications, the writer is of opinion that the corpus striatum intervenes between the hemisphere and the nerve nuclei in the medulla and cord in language as in the movements of the

limbs, and that groups of cells, representing words as mere motor processes, are formed or arranged in this ganglion, which are employed in the utterance of speech by higher centres in the cortex—by the third left frontal for ordinary intellectual expression—while they are evidently reached from some other centres and set in action as emotional manifestations, and possibly in yet another way when numbers are concerned.

Singular complications again sometimes arise when reading and writing are included in the consideration of affections of speech.

In a case which was made the subject of a communication to 'Brain,' the patient perused his newspaper regularly and with all the marks of intelligent interest. He understood it also, for he went to the sister in a state of great excitement to tell her of the failure of a firm with which he had business relations, carrying the paper in his hand and pointing out the announcement; and he could always find a given paragraph when asked to do so as a test. When, however, he was asked to read aloud the result was gibberish. The following passage was selected: —"You may receive a report from other sources of a supposed attack on a British Consul-General. The affair, however, is utterly unworthy of consideration. No outrage was even intended, and the report is due to misrepresentation of the facts. The Odessa line is again working properly." It was read slowly and in a jerky manner, as nearly as it could be taken down thus:—"So sur wisjee coz wenement ap ripsy fro fruz fenement wiz a secona coz foz no sophias a thu freehled pothy conollied. This affaise eh oh cout oh curly of consequences. Uce sudos val oh esses entain ah thee enepol a oh dee ah messequence oh coz foz. The assoil lens a puff pifl miss corres porety."

It was evidently an effort to read aloud, requiring close attention, and he read seriously and steadily, apparently unconscious of the absurdity of his utterances (probably because he was too intent on his task), till he was inter-

rupted by laughter which it was impossible to restrain, in which he usually joined.

When asked to copy a sentence he wrote the short words quickly and in a good hand, but a long word he took down slowly, letter by letter, in large schoolboy characters, usually accurately, but as he wrote each letter he named it aloud and always wrongly; he always, however, gave it the name of some letter or other, and not a name of his own invention.

In another case of a woman, the following verses taken from a book which the patient had been reading were taken down as read aloud:

First reading.

In litis deep its steepest tree
Under a spreading chestnut tree
In villi small and sound;
The village smithy stands;
The simist and the smallest man in this is he,
The smith a mighty man is he,
He that that sinneth abands,
With large and sinewy hands,
And the music of this biggy hands
And the muscles of his brawny arms
Those are this who are and is is abr.
Are strong as iron bands.
Are those who is imin abanded.

Second reading.

Then is speedily and cheery tree
The villi small and stern;
The smallest and the mightiest man is he
There is large and singing hands,
This is the smallest voice
Are arm of those who is the ini hands.

Another interesting case may be added where writing alone was affected. The patient, the editor of a paper, was seen with Mr. Archer, of Avenue Road, in January,

1881. A week or ten days before, during convalescence from bronchitis, he had found, while trying to make up some accounts, that he could not write what he wanted, and this continued. In all other respects his intellectual powers were unaffected. He was tested in this way. First he was asked to copy a few lines from a book, which he did rather badly, putting in two "that's." He was then requested to dictate a note to Mr. Archer begging him to make an appointment with me. This he did promptly and fluently as follows:—"My dear Sir, I shall feel much obliged if you will have the goodness to make an early appointment with Mr. Broadbent." Finally, he was asked to write a note to the same effect. He took a considerable time over it, with the following result:—"Thursday, 27th January. My dear Mr. Archbent, I dear Sir, I am thirty feel myself I think myself better though to some respects that I have respect in some respects someferred from those fured, &c. Yours very truly." He asked anxiously if it was right and if there was any mistake in it.

It is not proposed to discuss these affections of the faculty of reading and writing. They are briefly placed on record, together with the case which forms the principal subject of this paper, partly on account of their intrinsic interest, and partly as showing that the relation of cerebral lesions to affections of speech is a subject by no means exhausted, and as furnishing material which may contribute to the elucidation of the problem. It might, however, be shown how they fall into their places in the hypothetical scheme to which reference has been made.

EMBOLISM OF THE RIGHT MIDDLE CEREBRAL ARTERY,

PRODUCING

LEFT HEMIPLEGIA AND HEMIANÆSTHESIA:

ABSORPTION OF A LARGE PORTION OF THE RIGHT HEMISPHERE: DEATH SEVEN YEARS LATER.

BY

SEYMOUR J. SHARKEY, M.B.,

ASSISTANT PHYSICIAN TO ST. THOMAS'S HOSPITAL.

(Received May 13th—Read June 10th, 1884.)

E. J—, æt. 34, was admitted into Guy's Hospital on March 8th, 1876, under the care of Dr. Pavy, by whose kind permission I have made the following extract from the notes which were taken during her stay in the hospital:

She had enjoyed the best of health all her life, and had suffered from nothing more serious than some vaginal discharge and irregularity of menstruation. She had been married ten years, and had had neither children nor miscarriages. Three weeks before admission she was in her ordinary health, when suddenly she felt giddy, faint, and very sick. These symptoms passed off, but returned with equal suddenness on the following day. She likewise had twitching of the left arm and left side of the face, she

foamed at the mouth, was generally convulsed, and lost consciousness. She remained unconscious for four days, and on coming to herself found that she was completely paralysed and could feel nothing on the left side. She stated that little, if any, change had occurred in her condition up to the time of her admission into the hospital.

It was then found on examination that she was completely paralysed in the left arm and leg; the left angle of the mouth drooped, and the tongue was protruded to the left. On frowning and closing her eyes the muscles on the right acted much more energetically than those on the left, and when she whistled or laughed the mouth was drawn to the right.

In addition to this it was found that there was dimness of vision in the left eye almost amounting to complete blindness, though the patient said she saw perfectly before the attack. Hearing was good in the right ear, but almost absent in the left. Common sensation and taste existed on the right half of the tongue, but not on the left, and there was complete general anæsthesia of the left half of the body, affecting both the limbs and trunk. The sphincters were under the patient's control. The muscles of the eyeball were unaffected, the pupils equal, and the optic discs normal. Speech was but slightly altered. A systolic murmur was heard all over the cardiac area. Pulse was 76 and weak, and the temperature on both sides about equal. The urine was normal.

No marked improvement was noticed in her condition until March 15th, *i.e.* about four weeks after her illness commenced, when there was some evidence of returning sensation, the sight of the left eye was much improved, and the tongue diverged less to the left.

On March 20th she could taste and feel with either side of her tongue, but both sensations were less distinct in the left half than in the right.

On March 24th it was noted that she was irritable, stupid, remembering but little that was told her, but

having a much better memory for distant events. The anæsthesia of left side was less marked.

On April 1st she could execute some slight movements with her leg, and her general health was good.

On April 3rd, *i.e.* about six and a half weeks after the commencement of her illness, there was still more improvement in the power of the left leg, but the arm was as motionless as at first. Sight and hearing were completely restored.

After this changes occurred but very slowly. The arm was found on May 10th to have gained a little power, and sensation had quite returned all over the left side.

On May 31st she was getting about a little by herself, as the leg was much stronger, though the left arm was almost powerless.

On June 14th the patient returned home, about four months after the commencement of her illness, with the left arm almost useless, but with very fair power in the leg.

Seven years later, on May 7th, 1883, the patient, æt. 41, was admitted into St. Thomas's Hospital under the care of Dr. Harley, on account of difficulty of breathing and præcordial pain. She was thin, breathing rapidly, and expectorating whitish mucus. All over the cardiac area was heard a loud double murmur, most intense just to the left of the sternum. There were also signs of general bronchitis and of œdema of the base of the right lung. The liver was somewhat enlarged, the bowels constipated, and the urine contained albumen. The arteries were thick and the pulse 104.

Dr. Hadden, the registrar, made the following note:

"The fingers of the left hand are partially flexed on the palm and decidedly stiff. The skin over the backs of the fingers is whiter and more glistening than it is on the right hand. The grasp on the left is feeble and there is rigidity of the flexors of the forearm and arm. The tendon-reflexes are very brisk in the left, but are hardly obtainable in the right arm. The left leg is somewhat

rigid, the patella tendon-reflex brisk, and there is slight ankle-clonus. The tongue is protruded straight. Slight paresis is observed in the muscles on the left side of the face. There is no loss of sensation. On casual examination at the bedside the left eye appears to be sound, and there is no hemianopia or colour-blindness. Tested with a watch, hearing found to be sharp and equal on the two sides."

The patient died somewhat suddenly on May 17th.

The husband informed me that from the period of her attack, seven years before her death, she had been weak-minded and queer, doing unaccountable things such as she had never done before, but that she remained a good-tempered, hardworking, and affectionate wife.

On *post-mortem examination* marked aortic disease (ulcerative endocarditis) was found, and there was great cardiac hypertrophy, the heart weighing 17¼ oz. The lungs were hyperæmic and œdematous at the base, and the lower portion of the right was semisolid.

The liver showed slight congestion in the centres of the lobules ("nutmeg").

The kidneys weighed 11 oz., the capsule was thick and adherent, the substance very firm, and the surface granular.

The spleen was hard and its capsule thick.

Brain.—Great excess of subarachnoid fluid was present, which dripped freely away during the removal of the brain from the cranial cavity. In the region of the right temporo-sphenoidal lobe and inferior parietal convolutions the membranes were bagged out with clear serum, and there was evidently some defect in the substance of the brain at that spot. On rough examination it appeared that the greater part of the temporo-sphenoidal lobe, together with the convolutions which form the upper margin of the Sylvian fissure, had been absorbed, and that nothing remained but brownish-yellow *débris*. The uncinate and superior occipito-temporal gyri appeared to be normal. All the cerebral nerves appeared to be similar

on the two sides. The ordinary descending sclerosis of the postero-lateral region of the spinal cord and of the coloumn of Türck could be seen.

After the brain had been hardened in alcohol a more careful examination of it was made.

It was seen that the right hemisphere was very much smaller than the left, and the reduction was rather in bulk than in length; the extremities of the two hemispheres were nearly level. The transverse diameters, however, were very different. In the region of the fissure of Rolando the left hemisphere measured two and a half inches across, the right an inch and a half. The vertical measurement through the right hemisphere was also much less than that through the left.

The convolutions affected were those supplied by the Sylvian artery, viz. the inferior frontal, the external portion of the orbital, the lower half of the two central convolutions, the inferior parietal lobule, the convolutions of the island of Reil, the angular gyrus, the inferior and middle temporo-sphenoidal, and part of the inferior occipito-temporal convolutions.

These convolutions had completely disappeared, and a depressed, ill-defined, loose mass of a yellow colour remained. This consisted of the pia-arachnoid membrane, vessels, and shapeless granular *débris*, the relics of better things.

The two frontal convolutions which remained were smaller than those of the opposite side, but otherwise appeared to be unchanged. The upper halves of the two central or ascending convolutions were very much atrophied, but they were not completely destroyed like the other parts supplied by the middle cerebral artery.

The parieto-occipital fissure was three quarters of an inch in advance of its fellow, *i.e.* three quarters of an inch nearer the anterior extremity of the hemisphere, but the occipital lobe on the right was certainly not smaller than that on the left. It had apparently merely come forward to fill up the gap caused by the atrophy of the central

convolutions. The right half of the cerebellum appeared to be somewhat smaller than the left.

The optic thalamus on the right was much smaller in all dimensions than its fellow on the left. The anterior portion of the corpus striatum on the left was little if at all larger than that on the right, but this may have been due to a reduction in size of the left nucleus caudatus from disease, as there was an old cicatrix in it seen on section. The right lenticular nucleus was very markedly atrophied. Both corpora quadrigemina were smaller on the right side than on the left. The right half of the pons and the right pyramid were much reduced in size.

On examining a horizontal section passing through the island of Reil, corpus striatum, and optic thalamus on the two sides, and exposing the third and lateral ventricles, it was seen that the measurement from the intra-ventricular aspect of the basal ganglia to the outer surface of the convolutions of the island of Reil on the right was not much more than one third of that on the left, and that the intra-ventricular parts both of the corpus striatum and of the optic thalamus had suffered least.

The anterior cerebral artery on the right as far as the anterior communicating artery was larger than that on the left, beyond that point they were almost equal.

The Sylvian artery on the right and some of its branches contained rather hard red clot, some were empty. Most of those on the left side were empty.

Remarks.—This case is clearly one of embolism of the right middle cerebral artery occurring seven years before death, and resulting in subsequent absorption of those parts of the brain which are supplied by it. The points of greatest interest appear to be:

1. The complete absorption of so large a portion of one cerebral hemisphere without fatal results, the patient being left a useful member of society.

2. That although there was sudden loss both of motion and of special as well as of common sensation on the opposite side of the body, the loss of motion alone was

permanent; while the lesion of those parts, which the early symptoms proved to be sensory in their function, did not give rise to an incurable hemianæsthesia. On the contrary, the patient gradually and completely recovered common sensation, sight, hearing, taste, and smell, though loss of power remained.

The great destruction of the basal ganglia and internal capsule, as well as of many convolutions which are supposed to have special functions, renders this case useless for purposes of cortical localisation. Still some important conclusions may be drawn from it, viz.:

1. That if the angular gyrus and superior temporo-sphenoidal convolution be respectively connected with sight and hearing on the opposite side of the body, their destruction does not involve permanent loss of these special senses. Such an inference agrees with the results of experimental removal of the angular gyrus, an operation which has been found to produce crossed amblyopia, from which the animal soon recovers.

2. That each hemisphere *is* specially connected with the sight of the opposite eye. In a recent paper by Dr. Allen Starr,[1] on the visual area in the brain, the writer adopts the statement of Mauthner, made in 1881, that "there is no case well authenticated in which lesion of one hemisphere has produced blindness in the opposite eye;" and he concludes that "when a lesion of one hemisphere involves the optic fibres, at any point, partial blindness of both eyes, and not blindness of the opposite eye is produced."

Although I have seen cases where hemiplegia with hemianæsthesia including loss of sight and other special senses occurred on the same side of the body, and where a lesion of the opposite hemisphere appeared to be the cause of all, I have never before had the opportunity of verifying the fact at a post-mortem examination. As very careful notes of this case were made at Guy's, and on examination

[1] 'American Journal of the Medical Sciences,' January, 1884: abstract in 'Med. Times and Gazette,' March 22nd, 1884.

the left eye was found to be amblyopic and the right not, the conclusion that each hemisphere has a more special connection with the opposite retina than with that on the same side seems to me to be legitimate.

3. The case also supports the conclusion which other clinical and experimental observations very strongly suggest, that sensation—whether common or special—on each side of the body is not so indissolubly connected with certain definite areæ of the opposite hemisphere as is the case with motion. For the patient gradually recovered from her hemianæsthesia, notwithstanding the destruction of those regions, disease of which originally gave rise to it; a fact which shows that other parts, either of the same or of the opposite side of the brain, were capable of completely restoring the lost functions.

It may be said that this is not a necessary inference with regard to common sensation, since the hippocampal convolution was intact and a sufficient number of conducting fibres may have so far recovered that their functions could be re-established. Such an objection, however, is not valid as regards sight and hearing, since the convolutions specially connected with these senses were destroyed.

DESCRIPTION OF PLATES VIII AND IX.

Embolism of the Right Middle Cerebral Artery producing Left Hemiplegia and Hemianæsthesia (SEYMOUR J. SHARKEY).

PLATE VIII.

Fig. 1.—View of the Vertex of the Brain.
„ 2.—View of the Base of the Brain.

PLATE IX.

Fig. 1.—Lateral view of Left Hemisphere.
„ 2.—Lateral view of Right Hemisphere.

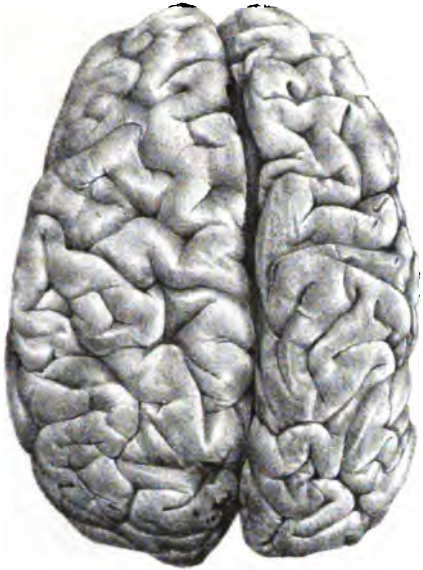


Fig. 1.

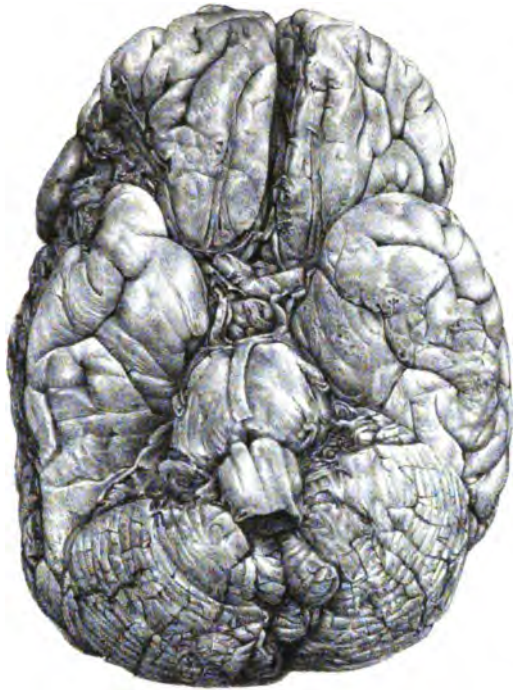


Fig. 2.

Mintern Bros. lith.

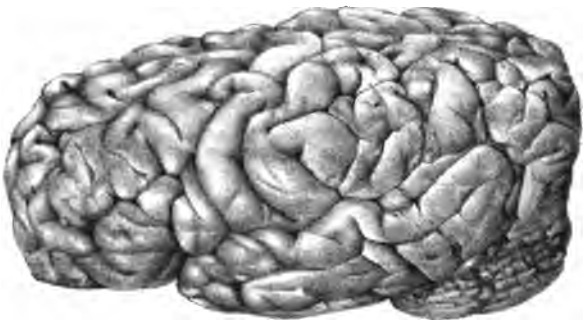


Fig. 1.

Finding the records of "Martha" in sufficiently good order from the beginning of 1881, Dr. Hurry made extracts of 32 cases of perimetritis and 21 of parametritis, 53 in all.

ON

ALBUMINURIA WITH PARAMETRITIS.

BY

J. MATTHEWS DUNCAN, M.D., F.R.S.,

OBSTETRIC PHYSICIAN TO, AND LECTURER ON MIDWIFERY AND DISEASES OF WOMEN AT, ST. BARTHOLOMEW'S HOSPITAL.

Received April 16th—Read June 10th, 1884.

It has repeatedly occurred to me, in taking care of cases of parametric inflammation, to be much more alarmed at the evidence of renal disorder offered by copious albuminuria than by the more striking parametric affection. In a recent example of psoas parametric abscess this occurred, the urine showing half albumen on boiling, and the woman having a puffy face and other symptoms attributable to the renal disturbance. In this prolonged case the abscess was opened in the right groin. It healed rapidly, and, as it healed, the albumen disappeared.

I therefore resolved, having the able and willing co-operation of my resident assistant at St. Bartholomew's Hospital, Dr. Hurry, to make use of my notes of cases of perimetritis and parametritis occurring there, with a view to elucidating the connection between these diseases and albuminuria.

Finding the records of "Martha" in sufficiently good order from the beginning of 1881, Dr. Hurry made extracts of 32 cases of perimetritis and 21 of parametritis, 53 in all.

In order to dispose at once of the 32 cases of perimetritis I shall now mention the remarkable restriction of the albuminuria to the parametritic cases. Among the 32 perimetritic cases there was not one of the kind of albuminuria of which I here treat. In 24 of the 32 cases no albumen was found. In 5 there was found only a trace, such as may be accounted for by the admixture of a small quantity of vaginal discharge. In the remaining 3 there was found albumen in considerable quantity, but also pus in quantity sufficient to account for the albumen; and this, in all these cases, without cystitic symptoms, the urine being acid, the pus being discharged into the bladder from a perimetric abscess.

It should be remarked that the diagnosis of perimetric from parametric phlegmon and abscess is not always quite secure; and it was premature to use this albuminuria as an important aid in the distinction. Parametric phlegmon or inflammatory swelling in the pelvic cellular tissue originating in uterine injury or disease, and parametric abscess or a collection of pus under the same conditions, are distinguished from perimetric phlegmon and abscess, or inflammation of and abscess within the peritoneum with great assurance. The indications are derived from the situation of the swelling, its relations, the direction of its spreading, the place of bursting, and the presence or absence of retraction of the thigh. In 10 of the 21 cases of parametritis the thigh was retracted. But although the diagnosis is in most cases secure I cannot ask for implicit faith in it as made in the 53 cases, to which reference is made in this paper. In all of them, however, the actual diagnosis was made and written before the investigation was thought of, and without using albuminuria as an element in it—a use which I shall, in future, feel justified in making.

In 11 of the 21 parametritic cases there was albumen in the urine. In 4 of these 11 cases there was also pus in the urine, and these 4 cases I exclude from the category available for the present discussion, the albu-

minuria being explained by the presence of pus: another case I exclude because the quantity of albumen was a trace. There are thus left 16 cases of parametritis without complication, and of these 16 cases 6 had albuminuria, or 37½ per cent.

In all of these 6 cases the albumen was in considerable quantity, from about one-tenth to above one half; in 1 case the urine on boiling was nearly solid.

In all the cases the urine had acid reaction; and the specific gravity varied from 1015 to 1030.

In 6 of the 16 cases the parametritis ended in abscess; and of the 6 cases with albuminuria 3 ended in abscess, the termination in abscess thus seeming to involve increased liability to albuminuria.

In all of the cases the albumen gradually diminished as convalescence from the parametritis was established. In 4 it completely disappeared before the patients left the hospital. In 2 (in 1 of which the albumen had been one quarter, and in the other the urine became nearly solid on boiling) the disappearance was very nearly completed at the time of leaving the hospital.

The 21 parametritic cases were, with one exception, puerperal. There is nothing extraordinary in this preponderance of puerperal cases; and the occurrence of albumen in the single non-puerperal case is important as indicating that the renal disorder is not confined to puerperal cases.

The albuminuria in these parametritic cases was too copious to be accounted for by vesical irritation; and besides, there was no vesical irritation in any of the cases, no cystitis, no symptoms of cystitis. The cases in which pus was found in the urine are, as already said, excluded from the category under discussion.

In a footnote (p. 23) to my translation of Braun's chapter on Uræmic Convulsions, published in 1857, it is said: "I have lately been able, in several cases, to attribute the occurrence of slight and temporary attacks of nephritis, in the early weeks of suckling, to exposure

to cold. These attacks have been, in some cases, accompanied by all the symptoms of a weid, and followed by slight œdema of the ankles and legs. In all the cases the urine presented fibrin cylinders under the microscope, and was in some proved to be albuminous." This extract I make because it might be supposed that the albuminuria and the parametric inflammation were both the results of the same cause in the cases under discussion. That it was so I know of nothing in the history of the cases to lead me to believe; and there are strong arguments against the view. Thus, the albuminuria of the parametritic cases was without casts except in one; the albuminuria occurred in the case that was not puerperal in its origin or history; the albuminuria was long continued, persisting during the parametritis; lastly, the albuminuria disappeared with the parametritis. It is, therefore, highly probable that the albuminuria is associated with the parametritis, as a consequence, and not caused by the cause of the parametritis.

This albuminuria cannot be confused with that resulting from prolonged suppuration; for in one half of the cases there was no suppuration, in the rest the suppuration was of short continuance, and in all the albuminuria diminished rapidly or disappeared with convalescence.

I am unable to suggest any sufficient reason for the association of copious albuminuria with this special inflammation, and, so far as I know, with no other. But the embryonic proximity of the kidneys and internal genital organs may be called to mind. It is also not rare to find the kidneys aching, and sometimes swollen and tender as well as aching, in menstruating women. Further, there is a well-known tendency of parametric inflammation to select, in spreading, the direction of the kidneys, and to produce inflammation of cellular tissue with or without abscess in the region of the psoas muscles and of the kidneys.

Whatever may be its explanation, it is important to recognise that in parametric inflammations there is a tendency to renal mischief as evidenced by albuminuria.

ON THE

WEIGHT OF THE THYROID BODY

IN

PERSONS DYING FROM VARIOUS CAUSES.

BY

STEPHEN MACKENZIE, M.D., F.R.C.P.,

PHYSICIAN TO, AND LECTURER ON MEDICINE AT, THE LONDON HOSPITAL.

(Received May 29th—Read June 10th, 1884.)

THE size of the thyroid body in disease has recently assumed considerable importance owing to a probable connection between atrophy and destruction of this body, and myxœdema and certain cretinoid conditions.

Comparatively few detailed observations appear to have been made as to the weight of the thyroid body in general diseases in which no obvious deviations from the normal were noticed in the organ during life.

In the years 1871 and 1872 I made a number of observations on the weight of the thyroid body of persons dying in the London Hospital. The observations were nearly consecutive in order, and made irrespective of the cause of death. They show also the age and sex of the patient, the weight and general nutrition of the body, and the cause of death.

The thyroid body was carefully dissected from its attachments, freed from adventitious tissues, and then weighed.

I think it better simply to record the observations arranged in a tabular form according to the ages, and to withhold any inferences until a larger series of cases allows of safer generalisation.

Table of the Weight of the Thyroid Body in Persons dying from various causes.

MALES.						FEMALES.				
	Age.	Nutrition of body.	Weight of body in lbs.	Weight of thyroid body. drm. gr.	Cause of death.	Age.	Nutrition of body.	Weight of body in lbs.	Weight of thyroid body. drm. gr.	Cause of death.
Under 5	1½	Normal	18	0 32	Acute tuberculosis.	4	Much wasted	—	0 45	Tubercular meningitis.
5 to 9	...	...	...	...	...	9	Œdematous	53	1 37	Acute nephritis.
10 to 14	11	Slightly wasted	65	2 19	Pyæmia.					
	12	Normal	70	3 44	Ruptured abdominal viscera.	12	Slightly wasted	59	2 3	Heart disease.
15 to 19	15	„	67	6 36	Accident.	18	Much wasted	103	5 46	„
						19	Normal	140	4 28	Peritonitis.
20 to 24	22	„	177	7 45	Fractured spine.	20	Much wasted	—	1 51	Heart disease.
	22	Slightly wasted	146	4 4	Rheumatic pericarditis.	23	Slightly wasted	—	4 24	Phthisis.
	22	Much wasted	155	3 9	Acute tuberculosis.					
	22	Normal	—	8 19	Enteric fever.					
	24	Stout	210	3 3	Heart disease.					
25 to 29	25	Slightly wasted	—	5 37	„	25	Much wasted	—	4 33	Myelitis.
	26	Normal	176	7 55	Fractured spine.	25	„	110	4 10	Acute phthisis.
	27	Slightly wasted	—	4 22	Stricture of urethra.	25	Stout	127	4 32	Heart disease.
	27	Much wasted	133	4 0	Pyæmia.					
	28	Slightly wasted	147	2 8	Bronchitis, emphysema.					
	29	Stout	170	3 24	Lithotomy.					
30 to 34	30	Much wasted	133	5 28	Cirrhosis of liver.					
	30	„	133	4 21	Tuberculosis.					
	31	Slightly wasted	182	5 18	Typhus.					
	31	Much wasted	134	3 47	Phthisis.					
	33	Emaciated	130	6 4	Abscess of shoulder.	32	Slightly wasted	140	4 11	Blood cyst of spleen.
	34	Much wasted	168	6 16	Aneurism of aorta.					
35 to 39	35	Slightly wasted	238	10 36	Cirrhosis of liver.					
	35	Normal	147	5 57	Poisoning by mercuric chloride.					
	36	Slightly wasted	126	3 56	Phthisis.					
	36	Emaciated	150	3 53	Heart disease.					
						38	Emaciated	114		Diabetes.

	41	Much wasted	147	3	59	Phthisis.					
	42	Normal	139	6	40	Bronchitis and emphysema.	42	Much wasted	119	2	46	Phthisis.
	42	Emaciated	119	3	50	Phthisis.						
	43	Slightly wasted	168	2	51	„	43	Emaciated	119	4	51	„
	43	„	210	9	16	Heart and kidney disease.						
45 to 49	45	Emaciated	126	3	17	Phthisis.	45	Stout	—	3	45	Strangulated hernia.
	45	Normal	154	4	38	Aneurism of aorta.						
	46	Much wasted	119	3	58	Phthisis.	46	Emaciated	112	2	35	Chronic Bright's disease.
	46	Œdematous	224	3	59	Chronic Bright's disease.						
	47	Slightly wasted	182	5	38	Heart disease.						
	48	Stout	202	4	25	Cerebral hæmorrhage.						
	49	Normal	—	5	11	Erysipelas.	49	Slightly wasted	139	2	50	Scirrhus of breast and lung.
	49	Much wasted	148	4	54	Chronic peritonitis.						
50 to 54	50	Normal	—	6	28	Shock.	50	Much wasted	133	2	45	Capillary bronchitis.
	52	„	—	4	14	Comp. fracture of leg.						
	52	„	168	7	20	Cancer of stomach.						
	54	Much wasted	148	3	46	Acute tuberculosis.						
	54	„	—	4	45	Cerebro-spinal meningitis.						
55 to 59	56	Normal	210	5	3	Bright's disease.						
	56	„	173	3	30	Pyæmia.						
	56	Much wasted	130	3	1	Chronic Bright's disease.						
	58	Emaciated	—	2	23	Stricture of œsophagus.						
	59	Much wasted	139	3	40	Bronchitis.						
60 to 64	60	Slightly wasted	—	6	9	Aneurism of aorta.	60	„	112	6	44	Chronic Bright's disease.
	61	Stout	184	2	23	Umbilical hernia.						
	62	Much wasted	147	5	3	Prostatic disease, bronchitis.						
	63	Slightly wasted	158	3	51	Cerebral hæmorrhage.						
65 to 69	68	Much wasted	127	3	46	Prostatic disease.						
	69	Emaciated	160	3	5	Stricture of œsophagus.						
70 to 74	70	Normal	217	9	41	Prostatic disease.						
	72	Much wasted	138	2	49	„						
	74	„	217	2	56	Erysipelas from injury.						
	74	Emaciated	130	3	41	Prostatic disease.						
75 to 79	79	Normal	133	3	26	Injury, run over.						

INDEX.

These Indices to the annual volumes are made on the same principle as, and are in continuation of, the General Index to the first fifty-three volumes of the 'Transactions.' They are inserted, as soon as printed, in the Library copy, where the entire Index to the current date may always be consulted.

ABSCESS, PSOAS, direct treatment of, with caries of the spine (F. Treves) 118

Details of the proposed operation, 115–118; account of three patients on whom it was performed, 119–123; pathological circumstances connected with the operation, 124.

Address (Annual) of the President (John Marshall, F.R.S.), March 1st, 1884 1

ALBUMINURIA with PARAMETRITIS (J. Matthews Duncan) 273

Records of 32 cases of perimetritis and 21 of parametritis made by Dr. Hurry, showing the restriction of albuminuria to the parametritic cases, 273–5.

ALCOHOLIC SPINAL PARALYSIS, on a form of (W. H. Broadbent) 133

Description of cases, and treatment, 133–140; characters and peculiarities of the disease, 141–144.

AMNESIA, on a particular form of:—loss of nouns (W. H. Broadbent) 249

Description of case, 249–51; post-mortem, 251–3; account of a second case, 253–5; analysis of language by disease of the brain, 254–5; part played by third left frontal gyrus and other parts of brain in articulate speech, 255–7; lesions of brain in first case described, 258–9; examples of affections of writing and speech, 262–4.

AMPUTATION, HIGH, for senile gangrene (J. Hutchinson) 91

Conditions under which the operation should be performed, 91–2; Mr. Garlike's case, 93; operation first proposed by Mr. James, of Exeter; description of cases, 94–99.

ANEURISM, SPONTANEOUS INGUINAL, in a boy aged twelve years; for which the external iliac artery was tied: with a record of all the other recorded cases of external aneurism in persons under twenty years of age (R. W. Parker) 51

Previous history of patient and condition on admission, 51–53; account of autopsy, 53–5; general remarks on the case, 55; table of fifteen cases, 57; mode of production of aneurism and its association with embolism; opinions of Dr. Kirkes, Sir W. Gull, Mr. Holmes, Dr. Church, Dr. Goodhart, Dr. Ponfick, &c., 60–65.

ARTERY, EXTERNAL ILIAC, ligature of, in case of spontaneous inguinal aneurism (R. W. Parker) . . 51

— RIGHT MIDDLE CEREBRAL, see *Embolism.*

ATROPHODERMA PIGMENTOSUM, see *Xeroderma pigmentosum.*

BISS, Cecil Y., M.A., M.D.
On the treatment of pus-secreting basic cavities of the lung by the method of paracentesis and free drainage . 217

BRAIN, ABSORPTION of a large portion of the right hemisphere, see *Embolism.*

— see *Cerebral Localisation.*

BROADBENT, W. H., M.D.
On a form of alcoholic spinal paralysis . . 133

On a particular form of amnesia: loss of nouns . 249

CARIES of the SPINE: direct treatment of psoas abscess with (F. Treves) 113

CAVITIES of the LUNG, see *Lung.*

CAYLEY, W., M.D., and *A. Pearce Gould.*
A case of gangrene of the lung following necrosis of the temporal bone the result of scarlet fever: treatment by drainage: recovery 209

CEREBRAL ARTERY, right middle, see *Embolism.*

CEREBRAL LOCALISATION, a review and forecast, being the Marshall Hall Prize Oration (David Ferrier) . 33

Comparison of researches of Marshall Hall and Flourens, 34; experiments by Prof. Goltz on brain of dog, 36; experiments on brains of monkeys, 37; conclusions drawn from preceding experiments, 39; cortical centres, 44; application of cerebral localisation to treatment of cerebral disease, 46.

CHAMPNEYS, Francis Henry, M.B.
Addendum to third communication on artificial respiration in stillborn children; (mediastinal emphysema and pneumothorax in connection with tracheotomy) . 101

— Fourth communication on artificial respiration in stillborn children; certain minor points . . 105

CHAVASSE, Thomas F., M.D.
Neurectomy of the second division of the fifth nerve . 145

CHILDREN, see *Stillborn Children*.

CROCKER, H. Radcliffe, M.D.
Three cases of xeroderma pigmentosum (Kaposi) or atrophoderma pigmentosum 169

DEFORMITY, SPINAL, three forms of (W. A. Lane) . 233

DIGITALIS GROUP, investigations into the action of the (S. Ringer and H. Sainsbury) . . . 67

Influence of digitalis on the circulation, 67–71; mode of experimentation with digitaline, barium chloride, strophanthus, scillitine, dyak poison, and convallamarin, 71–77; results of the experiments, 77–90.

DRAINAGE, treatment of a case of GANGRENE of the LUNG by; recovery (W. Cayley and A. P. Gould) . . 209

— FREE, on the treatment of pus-secreting basic CAVITIES of the LUNG by (C. Y. Biss) . . . 217

Druitt, Robert, obituary notice . . . 12

DUNCAN, J. Matthews, M.D., F.R.S.
On albuminuria with parametritis . . 273

EMBOLISM of the right middle CEREBRAL ARTERY, producing left hemiplegia and hemianæsthesia (S. J. Sharkey) 265

History of patient when under care of Dr. Pavy in 1876, 265–7; note by Dr. Hadden on her admission into St. Thomas's, 1883, 267–8; account of post-mortem, 268–70; remarks on the case and conclusions drawn from it, 270–2.

EMPHYSEMA, MEDIASTINAL, in connection with tracheotomy (F. H. Champneys) 101

Evans, George Henry, M.D., obituary notice . . 2

Fagge, Charles Hilton, M.D., obituary notice . . 9

Farr, William, M.D., *Honorary Fellow*, obituary notice . 18

FERRIER, David, M.D., LL.D., F.R.S.
Cerebral localisation, a review and forecast; being the Marshall Hall Prize Oration . . . 33

GANGRENE of the LUNG, treatment by drainage; recovery (W. Cayley and A. Pearce Gould) . . 209

— SENILE, on high amputation for (J. Hutchinson) . 91

GOULD, A. Pearce, see *Cayley and Gould*, case of gangrene of the lung.

Gully, James Manby, M.D., *of Malvern*, obituary notice . 7

Harding, John Fosse, obituary notice 8

HARLEY, John, M.D.
The pathology of myxœdema as illustrated in a typical case 189

Harper, Philip H., obituary notice 9

HEMIANÆSTHESIA, LEFT, produced by embolism of the right middle cerebral artery (S. J. Sharkey) . . 265

HEMIPLEGIA, LEFT, produced by embolism of the right middle cerebral artery (S. J. Sharkey) 265

HERMAN, G. Ernest, M.B.
An average or "composite" temperature chart of smallpox 205

HIGH AMPUTATION for SENILE GANGRENE, see *Amputation*.

Hudson, Robert Samuel, *of Redruth*, obituary notice . 2

HUTCHINSON, Jonathan, F.R.S.
On high amputation for senile gangrene . . 91

Iles, Francis Henry Wilson, M.D., *of Watford*, obituary notice 3

Jones, Thomas William, M.D., obituary notice . . 18

LANE, W. Arbuthnot.
Three forms of spinal deformity . . . 233

LIGATURE, see *Artery* (external iliac).

LOCALISATION, see *Cerebral Localisation*.

LUNG, GANGRENE of the, following necrosis of the temporal bone, the result of scarlet fever; treatment by drainage; recovery (W. Cayley and A. Pearce Gould) . 209
Description of patient, account of operation and progress of the case, 209—213; remarks, 214.

— pus-secreting basic cavities of, their treatment by paracentesis and free drainage (C. Y. Biss) . . 217
Pus-secreting basic cavities divided into three groups, 217–8; description of a case, with account of operation by Mr. Marshall, 218–226; post-mortem, 226; remarks on the diagnosis, 227–9; discussion of advisability of paracentesis, 229–231; literature of the subject, 231–2.

MACKENZIE, Stephen, M.D.
On the weight of the thyroid body in persons dying from various causes 277

Macnaught, John, M.D., *of Liverpool*, obituary notice . 6

MARSHALL, John, F.R.S.
Annual address as President, March 1st, 1884 . 1

Marshall Hall Prize Oration (David Ferrier) . . 33

MEDIASTINAL EMPHYSEMA, see *Emphysema*.

Meredyth, Adolphus William Leodore Colomiati, M.D., obituary notice 8

Montefiore, Nathaniel, obituary notice . . 14

MYXŒDEMA, the pathology of, as illustrated in a typical case (J. Harley) 189

Case originally under Dr. Ord and described by him ('Med.-Chir. Trans.,' lxi), 189; account of patient, 189–194; post-mortem and report by Dr. Hadden on cervical sympathetic, and some other of the tissues, 194–6; chemical examination of the tissues, 197–200; description of a second case, 201–3; relation of thyroid gland to myxœdema, 204.

NERVE, FIFTH, neurectomy of the second division of (T. F. Chavasse) 145

NEURECTOMY of the second division of the fifth nerve (T. F. Chavasse) 145

History of two cases and description of the operations, 145–9; operation first advocated by Carnochan, of New York, 149; results of twenty-two cases, 151; operations by Lücke, Lossen, Reyher, Nussbaum, Billroth, and Gerster, 152–3; table of twenty-four cases, 154–6.

NOUNS, loss of, a particular form of amnesia (W. H. Broadbent) 249

Obituary notices of deceased Fellows of the Society in 1883.

Druitt, Robert . . . 12
Evans, George Henry, M.D. 2
Fagge, Charles Hilton, M.D. 9
Farr, William, M.D., *Hon. Fellow* 18
Gully, James Manby, M.D., *of Malvern* . . . 7
Harding, John Fosse . . 8
Harper, Philip H. . . 9
Hudson, Robert Samuel, *of Redruth* . . . 2
Iles, Francis Henry Wilson, M.D., *of Watford* . . 3
Jones, Thomas William, M.D. . 18
Macnaught, John, M.D., *of Liverpool* 6
Meredyth, Adolphus William Leodore Colomiati, M.D. . 8
Montefiore, Nathaniel . . 14
Phillips, Richard . . . 14
Sims, James Marion, M.D. . 3
Stewart, Alexander Patrick, M.D. 14

PARACENTESIS, on the treatment of pus-secreting basic cavities of the lung by (C. Y. Biss) . . 217

PARALYSIS, ALCOHOLIC SPINAL, on a form of (W. H. Broadbent) 133

PARAMETRITIS, ALBUMINURIA with (J. Matthews Duncan) 273

PARKER, Robert William.

Case of spontaneous inguinal aneurism in a boy aged twelve years, for which the external iliac was tied: with a table of all the other recorded cases of external aneurism in persons under twenty years of age . . 51

Phillips, Richard, obituary notice . . . 14

PNEUMOTHORAX in connection with tracheotomy (F. H. Champneys) 101

PSOAS ABSCESS, see *Abscess.*

RESPIRATION, ARTIFICIAL, in stillborn children; addendum to third communication (mediastinal emphysema, and pneumothorax in connection with tracheotomy) (F. H. Champneys) 101

Statement by Dr. Angel Money as to occurrence of mediastinal emphysema and pneumothorax in post-mortems since the reading of Dr. Champney's paper ('Med.-Chir. Trans.,' lxv).

— fourth communication:—certain minor points (F. H. Champneys) 105

Effect of air in the abdominal viscera on the amount capable of being drawn into the lungs, 105; methods to prevent entrance of air into stomach in mouth-to-mouth inflation, 107; patency of upper part of air-passages, 108; progressive signs of returning to life, 111; works quoted, 112.

RINGER, Sidney, M.D., and *Harrington SAINSBURY*, M.D.

Investigations into the action of the digitalis group . 67

SAINSBURY, Harrington, M.D., see *Ringer and Sainsbury* (action of the digitalis group).

SENILE GANGRENE, see *Gangrene.*

SHARKEY, Seymour J., M.B.

Embolism of the right middle cerebral artery, producing left hemiplegia and hemianæsthesia; absorption of a large portion of the right hemisphere; death seven years later 265

Sims, James Marion, M.D., obituary notice . . 3

SMALLPOX, an average or "composite" temperature chart of (G. E. Herman) 205

SPINA BIFIDA, LARGE, radical cure of, in an adult (W. Whitehead) 127

Description of the patient, 127–129; account of the operation, 130; conclusions drawn from the case, 132.

SPINAL CORD, see *Alcoholic spinal paralysis.*

SPINAL DEFORMITY, three forms of (W. A. Lane) . 233

Description of spinal deformity among men who carry heavy burdens (1) on their right shoulder, 234; (2) on their back, 239, and (3) on their heads, 241.

SPINE, CARIES of the, direct treatment of psoas abscess with (F. Treves) 113

Stewart, Alexander Patrick, M.D., obituary notice . 14

STILLBORN CHILDREN, on the value of systematic examination of (J. B. Sutton) 157

Recent endocarditis in a fœtus, mother's heart normal, 158; constriction of stomach, with intestine terminating in a cul-de-sac, 159–61; spinal curvature, 161–2; abnormal conditions of the kidneys, 162–3; deficiency of the diaphragm, 163; cleft-palate; meningo-encephalocele, 163–4.

— ARTIFICIAL RESPIRATION in, see *Respiration* (artificial).

SUTTON, John Bland.

On the value of systematic examination of stillborn children 157

TEMPERATURE CHART, an average or "composite," of smallpox (G. E. Herman) 205

Description of four charts, 205–7.

THYROID BODY, on the weight of the, in persons dying from various causes (S. Mackenzie) 277

Table of cases arranged according to ages, 278–9.

TRACHEOTOMY, mediastinal emphysema and pneumothorax in connection with (F. H. Champneys) . . 101

TREVES, Frederick.

The direct treatment of psoas abscess with caries of the spine 113

Wheatley, Benjamin Robert, *Resident Librarian*, obituary notice 21

WHITEHEAD, Walter.

Radical cure of a large spina bifida in an adult . 127

XERODERMA PIGMENTOSUM (Kaposi) or Atrophoderma pigmentosum, three cases of (H. R. Crocker) . 169

Disease first described by Hebra, 169; description of three cases, 170–176; etiology and symptoms, 176–9; description of the tumours, 179; diagnosis, prognosis, treatment, and pathology, 180–2; "atrophoderma" suggested in place of "xeroderma," 183; table of 34 cases, 184–7.

PRINTED BY J. E. ADLARD, BARTHOLOMEW CLOSE.